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THE ETIOLOGY OF GASTRIC AND DUODENAL ULCER[1]

EXPERIMENTAL STUDIES

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DURING the past 15 years there has been a renewed clinical interest in the problems presented by the etiology and pathogenesis of gastric ulcer and a large amount of excellent experimental work has been done which has greatly clarified already existing ideas concerning the significance of various factors in the cause of the disease. Perhaps the most important development during this period has been the successful production of chronic ulcer in the experimental animal. The early literature is replete with unsuccessful attempts to produce a chronic progressive lesion in the gastric or duodenal mucosa of dogs. This effort has served, however, to make evident the great capacity of the gastric mucosa of these animals to heal in the presence of the usual gastric content and after the most extensive mechanical and chemical traumas. Bolton (1913), Friedman and Hamburger (1914), Dragstedt and Vaughn (1924), Shapiro and Ivy (1926), Wolfer (1926), and others have produced chronic gastric ulcers in dogs by methods which are chiefly serviceable in determining the subsequent effect of these ulcers on the secretory and motor function of the stomach, but are of less significance in determining the cause of the spontaneous lesion. Exalto (1911) and especially Mann and his associates (1923) must be credited with being the first to develop methods which regularly lead to the production of chronic ulcers without the use of external destructive agencies. Largely as a result of the experiments of Mann and his associates in this country, the theory that ulcer is due primarily to the corrosive and digestant action of the pepsin-hydrochloric acid of the gastric juice has received a renewed emphasis and for the first time definite experimental support.

In this connection, however, and in spite of the evidence presented in this paper, it must be recognized that in dealing with the clinical problem many diverse agencies may produce an acute lesion in the human stomach. Such a lesion is not ordinarily under optimum environmental conditions for healing and we may list for subsequent examination those more obvious factors which might delay healing and induce chronicity. First, in otherwise healthy individuals it seems likely that the exposure of the acute lesion to the corrosive action of the gastric content would delay healing. In this connection the time of such exposure and the concentration of free acid and of pepsin in the gastric content should be of great significance. Second, comes the almost ceaseless motility of the stomach and especially of the pyloric region where chronic ulcers are so prone to develop. Here also the increased motility associated with pylorospasm and retention demands consideration. Third, the possible mechanical effect of coarse

[1]This work has been conducted under a grant from the Douglas Smith Foundation for Medical Research of The University of Chicago.

food particles pressed into an acute ulcer by the digestive motility of the stomach is of significance. In addition to these factors operative in healthy persons we must bear in mind a possible generalized decrease in the rate of healing or in the resistance of the gastric or duodenal mucosa to the digestant action of the gastric juice present in individuals rendered cachectic by anæmia, food deficiencies, endocrine disturbances, etc., through local vascular disease or thrombosis of the gastric blood vessels, decrease in the amount of gastric mucus, etc. Since chronic gastric ulcer is predominantly a disease of young and otherwise healthy adults, it seems improbable that these latter factors play any large rôle in the disease.

The present experiments were designed to determine in so far as possible the rôle of the chemical action of the gastric juice in the cause of ulcer and its relation to the chronicity of these lesions.

Interest in this problem has centered about the pepsin and hydrochloric acid rather than other constituents of the juice because of their great capacity to hydrolyze proteins. The question has been raised, "Why does the stomach not digest itself?" It has been commonly assumed that the mucosa lining the gastric wall has some specific resistance to such digestion not possessed by other living tissues and entirely absent in dead protein. An attempt was made by Dragstedt and Vaughn (1924) to secure experimental evidence regarding the resistance of various tissues to gastric digestion. In their experiments, large windows were produced in the stomach of dogs and into these defects were carefully sutured segments of duodenum, jejunum, ileum, colon, and such organs as spleen and kidney. In no case were these tissues digested away. The exposed surfaces of the spleen and kidney were soon covered by a layer of newly formed gastric mucosa while the mucosa of the duodenal and intestinal implants remained entirely normal for periods of at least 9 months. It is thus quite evident that there exists a widespread resistance to the digestant action of the normal gastric content on the part of tissues and organs whose blood supply is not interfered with. It should be emphasized that this experiment yields data only on the resistance of the tissues to the normal gastric content but not to pure gastric juice.

The experience of innumerable physicians during the past 50 years has established the fact that the free hydrochloric acid in the gastric content aspirated 1 hour after the ingestion of an Ewald meal varies between 30 and 60 clinical units in the case of normal individuals. Data obtained by the method of fractional gastric analysis advocated by Rehfuss and his associates have in general confirmed this belief. On the other hand, it has been equally well established that the concentration of free acid in pure gastric juice, such as may be obtained from isolated portions of the stomach as in the experiments of Heidenhain and Pawlow, is much greater than this. The average free hydrochloric acid in the uncontaminated gastric secretion yielded by the completely isolated dog's stomach in the experiments of Dragstedt and Ellis (1930) varied between 0.35 and 0.49 per cent, the latter figure being by far the more common finding. These values correspond to approximately 100 and 135 clinical units. A similar figure (0.40 to 0.50 per cent free hydrochloric acid) has been given for pure unmixed gastric juice in man (Carlson, 1923).

If it be conceded that it has been difficult or impossible to demonstrate any deterrant effect on the healing of acute lesions in the stomach of experimental animals due to the corrosive chemical action of the normal gastric content, the question naturally arises, may not *pure gastric juice* with its *higher free acid* exert such an effect? As a corollary to this we may perhaps profitably inquire into the factors which normally reduce the acidity of pure juice to that of the normal gastric content. Those that come readily to mind may be listed as the neutralizing effect of food and swallowed saliva, mucus secreted in the œsophagus, fundus, and particularly in the pyloric antrum of the stomach, fixed base secreted chiefly in the pyloric antrum, and regurgitated alkaline juices from the upper duodenum. Does a defect in this neutralizing mechanism or any part of it account for the fact that a majority of patients with duodena

TABLE I.—INTESTINAL ULCER AFTER IMPLANTATION OF STOMACH POUCHES

The incidence of chronic ulcer in the intestine following the implantation of stomach pouches (experimental diverticulum ulcers). Pawlow pouches were used in all cases except Dogs 5 and 6, in which Heidenhain pouches were employed

Dog No.	Size of pouch	Part of intestine used	Length of experiment	Fate of animal	Description of ulcer	Condition of animal at death
1	Small	Ileum	80 days	Sacrificed	2 x 3 cm.	Good. No weight loss
2	Small	Ileum	60 days	Died of distemper	2 x 1.5 cm. Subacute	Poor
3	Small	Ileum	77 days	Sacrificed	4 x 3 cm. Chronic	Fair. 20% weight loss
4	Small	Ileum	77 days	Sacrificed	2 x 3 cm. Chronic	Fair. 15% weight loss
5	Small	Ileum	34 days	Peritonitis	4 cm. diameter, 2 cm. deep. Perforated	Good. No weight loss
6	Small	Ileum	91 days	Sacrificed	1.5 cm. diameter, 0.5 cm. deep. Chronic	Poor
7	Large	Jejunum	29 days	Peritonitis	Size half hen egg. Perforated	Poor
8	Large	Jejunum	51 days	Hæmorrhage	4 cm. wide, 4 cm. deep	Good. 10% weight loss
9	Large	Jejunum	92 days	Intussusception	No ulcer	Very poor
10	Large	Jejunum	86 days	Peritonitis	Extensive destruction intestinal wall	Good. No weight loss
11	Large	Jejunum	24 days	Accidentally killed	3 cm. diameter. Chronic	Good. No weight loss
12	Large	Jejunum	83 days	Peritonitis	Crater, size goose-egg	Very poor
13	Large	Jejunum	98 days	Sacrificed	4 cm. wide, 4 cm. deep. Crater	Poor
14	Large	Jejunum	103 days	Evisceration	No ulcer	Good. No weight loss
15	Large	Jejunum	93 days	Not killed	2 cm. diameter. Chronic	Good. No weight loss
16	Large	Jejunum	22 days	Peritonitis	2 x 4 cm. Perforated	Fair. 10% weight loss
17	Large	Jejunum	52 days	Peritonitis	4 x 3 x 3 cm. Chronic	Poor. 30% weight loss
18	Large	Jejunum	14 days	Peritonitis	1.5 cm. diameter	Good. No weight loss
19	Large	Jejunum	160 days	Peritonitis	Chronic 5 x 4 cm.	Poor. 50% weight loss

or gastric ulcer have a gastric content of higher than normal acidity and one which approaches the acidity of pure juice?

THE PRODUCTION OF CHRONIC ULCER BY PURE GASTRIC JUICE

a. *Meckel's diverticulum ulcer.* Perhaps the most striking evidence to be obtained from human pathology in support of the view that the chemical action of gastric juice may produce a chronic ulcer is the occurrence of such a lesion in the mucosa of the ileum adjacent to the entrance of Meckel's diverticulum. Aschner and Karelitz (1930) and Lindau and Wulff (1931) have summarized the clinical literature bearing on this problem and have pointed out that in those cases in which ulcer has been present, islands of heterotopic gastric mucosa, histologically similar to that in the fundus of the stomach, have been almost invariably found in the diverticulum. Additional proof that this heterotopic gastric mucosa is functional has been cited in the occasional persistence of the vitelline duct as a fistula opening at the umbilicus and discharging an acid proteolytic secretion capable of corroding the skin. Both pepsin and free hydrochloric acid have been detected in this secretion (Lindau and Wulff), and the amount of secretion has been found to increase greatly when food is taken in the stomach. This is undoubtedly due to the stimulating effect of gastrin bodies discharged into the general circulation during gastric digestion since secretory nerves to these islands of gastric tissue have not been found. In this connection it should be noted that Ivy and Farrell (1925) autotransplanted small pouches of the fundic portion of the stomach subcutaneously in dogs and observed that following a meal the transplant secreted acid. All nervous connections between the transplant and the main stomach had been severed. The Meckel's diverticulum ulcer invariably occurs adjacent to the heterotopic gastric mucosa but always involves only the mucosa of the ileum.

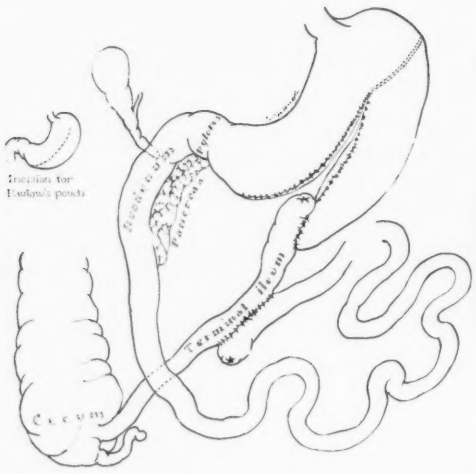

Fig. 1. Diagram showing the method of implantation of the Pawlow accessory stomach into the ileum for the experimental production of "Meckel's diverticulum ulcer."

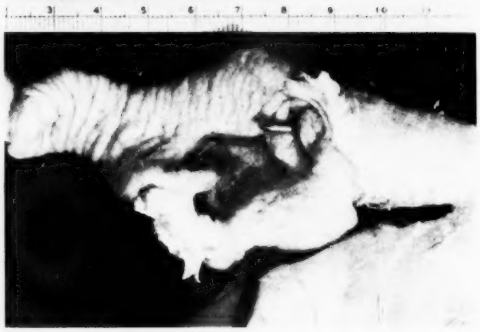

Fig. 2. "Experimental Meckel's diverticulum ulcer" of the ileum in Dog 4, Table I. The gastric pouch is to the right of the picture. A probe protrudes from the opening of this pouch in the ileum. Note the large size of the ulcer and its proximity to the gastric mucosa.

This fact suggests that the ileal mucosa is more susceptible to the chemical action of gastric juice than is gastric mucosa, a point to be elaborated upon later in the discussion. The ulcer appears early in life, usually within the first 18 months, and repeated hæmorrhages and occasionally perforation occur.

Because of the theoretical importance of these clinical observations and the possibility that the mucous membrane of the dog might behave somewhat differently, we have at the suggestion of Dr. D. B. Phemister performed experiments designed to imitate so far as possible the situation found in Meckel's diverticulum ulcer.[1]

Experimental procedure. Healthy dogs were secured and all operations were performed under complete ether anæsthesia with the usual aseptic precautions. A small Pawlow pouch, 2 by 3 by 8 centimeters, was made from the greater curvature of the stomach in the region of the fundus. A segment of ileum, about 30 cubic centimeters proximal to the ileocæcal valve, was then selected, the intestine divided, and the distal ileum united to the open end of the Pawlow pouch. The continuity of the intestine was then re-established by anastomosing the proximal ileum to the distal intestine about 15 centimeters below its attachment to the gastric pouch (Fig. 1). Particular care was taken not to bruise the tissue or to damage its blood supply. No clamps were applied and only chromic catgut (No. 00) was used for the anastomoses. In a few of the experiments a pouch, the nervous connections of which had been first severed (Heidenhain pouch), was used for anastomosis with the lower intestine. After the results of these experiments were determined, the operations were repeated on other animals, but in this case a very large Pawlow pouch was made (approximately ½ to ⅔ of the entire stomach) and its open end anastomosed to different regions of the jejunum, in some cases as far proximal as the ligament of Treitz.

The animals recovered from the operations promptly. They were given saline intravenously but nothing by mouth for the first 4 to 5 days. Following this they were given the regular stock diet of the laboratory, consisting of ground meat, bread, carrots, and other vegetables and were fed once a day. Those that survived for as long as 2 to 3 months were usually subjected to a second laparotomy and the condition of the intestine determined by inspection.

The results of the experiments are summarized in Table I. Of the 19 animals operated upon, 8 died of peritonitis following per-

[1] A preliminary report of this work was published in the Proceedings of the Society for Experimental Biology and Medicine, 1931, xxviii, 960.

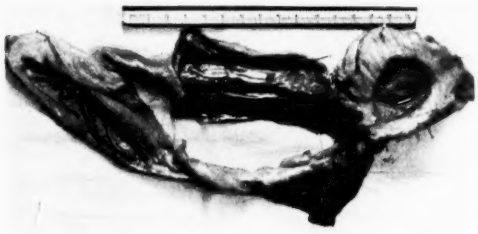

Fig. 3. "Experimental Meckel's diverticulum ulcer" in the jejunum of Dog 8, Table I. The opened gastric pouch is above and to the left of the ulcer. Note the large size of this ulcer, its sharp margins, and the persistence of a narrow zone of jejunal mucosa between the ulcer and the gastric mucosa.

foration of an intestinal ulcer, 6 were sacrificed when they became markedly cachectic, while the 5 remaining were in excellent condition at the time they were etherized for examination. Seventeen of the 19 animals had developed large typical chronic ulcers in the intestine adjacent to the anastomosis with the gastric pouch. In the cases in which the isolated gastric pouch was implanted into the ileum (6 dogs), chronic ulcers appeared in all, an incidence of 100 per cent, whereas when the implantation was made in the jejunum even though a much larger pouch was used, the incidence of ulcer was less (11 ulcers in 13 animals operated upon or 85 per cent). In many cases (roughly ⅓ of the total) the ulcer seemed to have little deleterious effect upon the general condition of the animal until a sudden perforation or exploratory laparotomy revealed its presence. The shortest time elapsing between the date of operation and discovery of the ulcer was 14 days, the longest 160 days, and the average, 67 days.

It is significant that the ulcers always developed in the intestinal wall adjacent to the line of union with the gastric mucosa (Figs. 2 and 3). In most cases a narrow line of intestinal mucosa was visible between the ulcer and the nearest gastric mucosa. The latter was never involved. The ulcers presented the same clean, punched-out, "digested" appearance characteristic of the lesion in man. Their size was often enormous, and in several a crater large enough to contain an average size hen's egg was present. The floor of such an ulcer was composed of hyper-

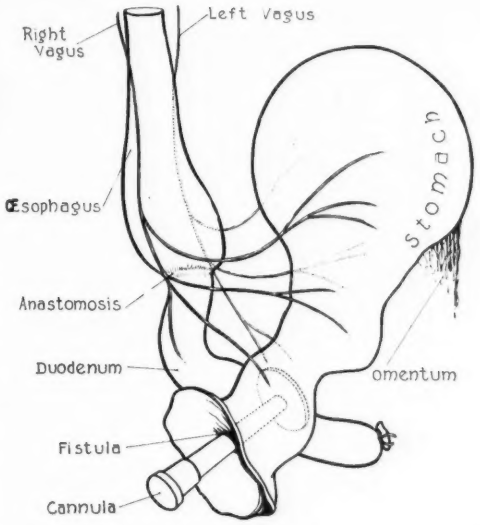

Fig. 4. Diagram showing the method of preparation of the isolated stomach with vagus innervation intact. (From Dragstedt and Ellis, Am. J. Physiol., 1930, xciii, 407.)

plastic fibrous connective tissue, the remains of an inflamed, indurated omentum, loops of neighboring intestine, mesentery, and in some cases organs such as the pancreas or liver. All were combined to form a large indurated inflammatory mass, requiring very careful dissection.

Microscopic examination of the intestinal mucosa in the immediate neighborhood of the ulcer presented evidence of inflammation, such as round cell infiltration, suggestive of the so called Konjetzny gastritis.

The successful production of chronic perforating ulcer in the ileum of dogs under conditions resembling those in the Meckel's diverticulum ulcer in man indicates that the susceptibility of the two species to the development of this disease may not be greatly different under comparable conditions. It seems probable that we may disregard the factor of operative trauma as of any great significance in the genesis of the experimental ulcer. In a few cases the region of the anastomoses was examined at a second laparotomy from 7 to 14 days after the first operation, and in each case healing was complete or progressing satisfactorily without evidence of ulcer. Further-

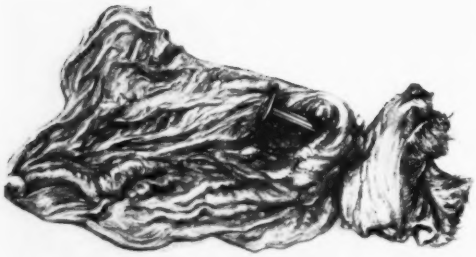

Fig. 5. Drawing showing the formation of a large, chronic, progressive ulcer in the isolated stomach.

Fig. 6. Chronic perforating ulcer occurring spontaneously in a very large Pawlow pouch. The mucosa of the lesser curvature or *Magenstrasse* shown below to the right and that of the first part of the duodenum to the left are normal.

more, the almost invariable persistence of a strip of intestinal mucosa between the ulcer and the line of anastomosis suggests that the probable vascular damage to the intestine as a result of incision and subsequent suture was not of paramount importance in the ulcer formation. In man, of course, there can be no question of trauma or vascular injury, and we must attribute the ulcer to the destructive effect of the gastric juice. In both cases it is probably of considerable importance that the gastric juice enters an ileum empty of food or secretion and consequently its free acid is little reduced. Secretion of gastric juice in the Pawlow pouch begins with the entrance of food in the mouth as a result of reflex stimulation of secretory nerves in the vagi and accordingly its pepsin hydrochloric acid comes in contact with the mucosa of the ileum long before food reaches this level and could possibly exert a neutralizing effect. Even in the experiments with the Heidenhain pouches and in the clinical cases it is likely that the hormone stimulation of the gastric mucosa produces a secretion before food reaches the ileum. At all events the production of intestinal ulcers by implants of gastric mucosa, completely separated from all connections with the central nervous system, speaks volumes against the rather fanciful idea that the latter plays a rôle in the proximal cause of the lesions. As noted above the incidence of ulcer in the intestine was 100 per cent when the gastric pouch (with or without nerves) was implanted into the ileum but only 85 per cent when the implantation was made in the jejunum. This may be interpreted to indicate a greater resistance on the part of the jejunal mucosa or it may be due to the earlier appearance of the chyme and neutralizing secretions of the upper duodenum.

Fig. 7. Photograph showing extensive digestion of the mesentery following rupture of a chronic ulcer in a large Pawlow pouch permitting several hundred cubic centimeters of very acid gastric juice to escape into the peritoneal cavity.

The absence of ulcer formation in segments of jejunum or ileum implanted into the stomach as in the experiments of Dragstedt and Vaughn (1924) and the uniform occurrence of such lesions in the intestine when segments of stomach are implanted as in the experiments described, are striking and, at first glance contradictory phenomena. The

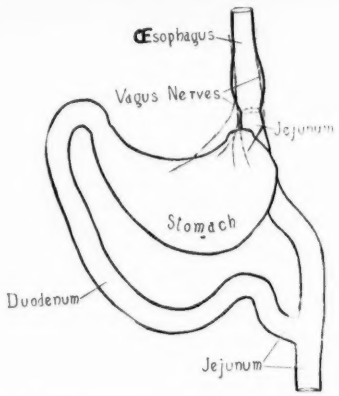

Fig. 8. Diagram showing the type of preparation used to determine the effect of gastric secretion in the stomach empty of food on the occurrence of ulceration of the intact gastric mucosa.

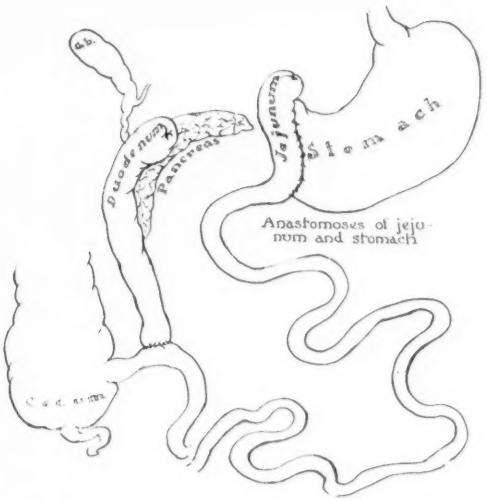

Fig. 9. Diagram showing the author's modification of the experiment of Mann for the production of jejunal ulcer by "surgical duodenal drainage." In spite of the resection of the pyloric antrum and the large anastomosis, chronic ulcers developed in 100 per cent of cases.

essential difference in the two experiments lies probably in the removal of the entire neutralizing mechanism in the latter case so that the intestinal mucosa is exposed to the acid pepsin concentration of *pure fundus secretion* whereas when the intestine is implanted into the stomach it is exposed only to the acid-pepsin concentration of the usual gastric content, i.e., after dilution and partial neutralization by the mechanisms suggested in the introduction.

b. *Occurrence of chronic perforating ulcer in the isolated stomach.* It is noteworthy in the experiments described in the preceding section that in no instance did ulcers appear in the gastric pouch, whereas in a very large proportion of cases, large, chronic, progressive ulcers developed in the mucosa of the ileum and jejunum. The operative trauma to the gastric mucosa was obviously greater and it must have been exposed to as high or a higher concentration of pepsin hydrochloric acid than the intestine. These facts suggest that the

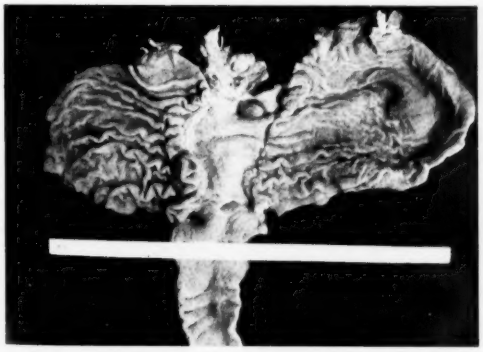

Fig. 10. Photograph showing the formation of a jejunal ulcer following the type of operation illustrated in Figure 9.

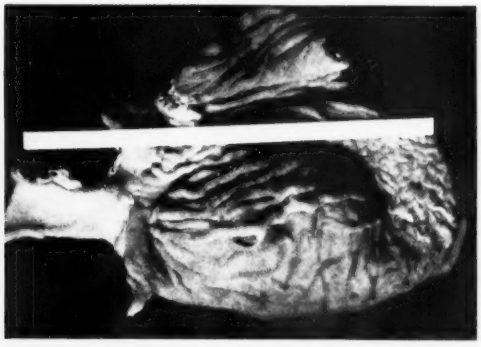

Fig. 11. Photograph showing the formation of a small jejunal ulcer near the suture line and three small gastric ulcers following an operation as in Figure 9. These gastric ulcers are very unusual.

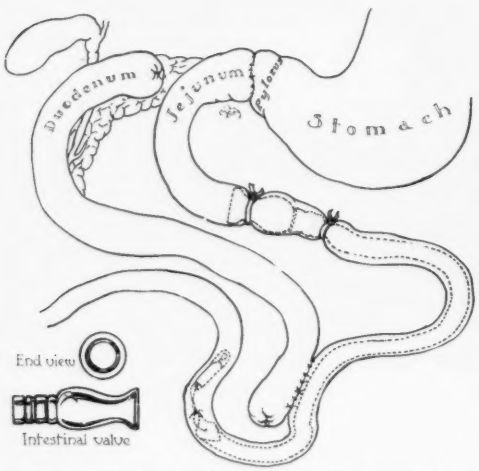

Fig. 12. Diagram illustrating the author's method for producing jejunal ulcers by "surgical duodenal drainage" with high implantation of the duodenum. Regurgitation of duodenal juices in the jejunum is prevented by the valve.

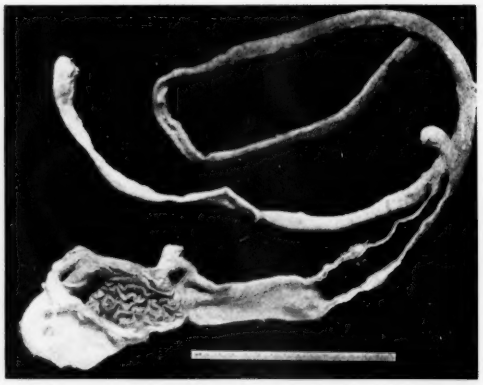

Fig. 13. Photograph showing typical jejunal ulcers in Dog 1, Table II, after operation illustrated in Figure 12. In this case an end-to-side anastomosis was made between the stomach and jejunum.

gastric mucosa does actually possess a greater resistance to the digestant action of pure gastric juice than is present in the mucosa of the intestine lower down. If pure, unneutralized gastric juice is able to produce a chronic ulcer in the stomach, it would appear that the optimum conditions for such a lesion should exist in the so called isolated stomach. Frémont (1895) made a brief allusion to some experiments performed on dogs in which he had isolated the stomach, suturing the duodenum to the œsophagus and collecting the gastric secretion by means of a fistula. Inasmuch as he states that such a stomach to which the vagi have been cut secretes a fluid containing no acid and is non-digestive, we may question whether he actually secured a good preparation. Lim, Ivy, and McCarthy (1925), however, isolated the entire stomach of dogs, using the method described by Frémont, and Ivy has kept such animals alive for a number of years. Although the vagus nerves to these isolated stomachs have been cut they secrete a highly acid proteolytic juice. In no case has Ivy (1931) observed an ulcer in such a stomach. It should be noted, however, that in all these experiments the gastric juice has been permitted to flow away from the stomach by way of a fistula almost as soon as it is secreted so that there is at no time a prolonged contact of the active juice with the mucosa. It is likely that this fact is of decisive importance. L. R. Dragstedt and Ellis (1930) described a method for preparing an isolated stomach in the dog, leaving the vagus innervation intact and making a fistula with a metal cannula, by means of which it is possible to retain pure gastric juice in the stomach at will (see Fig. 4). During the past 5 years a number of such animals have been kept in the laboratory. For the most part gastric juice has been permitted to flow out of the fistula into a collecting rubber bag so that to date the full advantage of the method for determining the effect of pure gastric juice on gastric mucosa has not been obtained. However, in one case a large typical chronic ulcer (see Fig. 5) was found in the isolated stomach. As will be noted from the illustration this lesion occurred close to the entrance of the cannula so that we cannot exclude this mechanical factor in the etiology. However, when such a cannula is placed in the normal intact stomach or intestine it has never produced such a defect.

c. *Occurrence of a chronic perforating ulcer in a Pawlow accessory stomach.* A small isolated accessory stomach made from the fundus, after the method of Pawlow, has been widely used in many physiological laboratories during the past 30 years for the study of

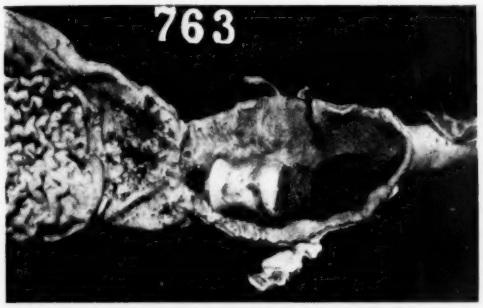

Fig. 14. Photograph showing a chronic perforating jejunal ulcer in Dog 5, Table II, after an operation as illustrated in Figure 12. The valve has been partly withdrawn to show its shape and condition after having been in place 54 days. Its previous location is indicated by the tag of omentum adherent to the intestine about 18 centimeters from the ulcer.

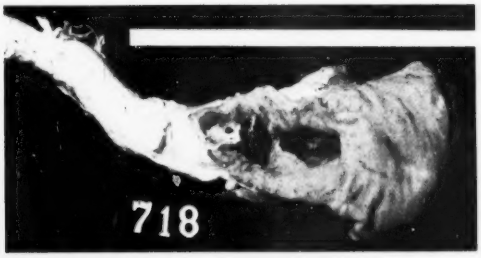

Fig. 15. Photograph showing a large perforating jejunal ulcer in Dog 7, Table II, after an operation as illustrated in Figure 12. The edge of the ulcer is exactly at the line of anastomosis with the stomach. The floor of the ulcer is made up of liver, which is partially eroded.

the physiology of gastric secretion. To our knowledge no one has to date reported the spontaneous occurrence of an ulcer in such a pouch. As noted in the preceding section, this may be due to the fact that the gastric juice is commonly promptly drained away by the fistula and so does not remain in contact with the gastric mucosa for any appreciable length of time. However, in 1917, one of us (L.R.D.) tried to prepare Pawlow pouches in dogs in such a way that the gastric secretion should be retained in the pouch, by implanting the fistulous opening in the skin of the lateral abdominal wall, instead of on the ventral surface. Spontaneous ulcers did not occur in these pouches and acute lesions produced by silver nitrate seemed to heal as readily as in control dogs where the accessory stomach was continually drained. An examination of the data respecting the acidity of the gastric juice from these pouches, however, suggests an explanation for these negative results. The free hydrochloric acid varied between 0.00 and 0.109 per cent with an average of about 0.045 per cent. This is far below the acidity of the juice from the isolated entire stomach (as noted above) and is within the range of acidity of the normal gastric content, which Dragstedt and Vaughn (1924) found to be relatively innocuous even to the exposed spleen. By means of a technique modified considerably from that given by Pawlow, we have been able to construct an isolated accessory stomach containing from $\frac{2}{3}$ to $\frac{3}{4}$ of the fundus, leaving only a channel along the lesser curvature not larger in diameter than the duodenum below. Such a large accessory stomach may secrete from 800 to 2,800 cubic centimeters of gastric juice in 24 hours with a free acidity ranging between 0.25 and 0.49 per cent (Dragstedt and Ellis, 1930). In one such preparation a large chronic ulcer (see Fig. 6) formed in the Pawlow pouch and ruptured 4 months after the original operation, producing a fatal peritonitis. A striking feature of such a peritonitis which results in the liberation of an exceedingly active secretion into the general peritoneal cavity is illustrated in Figure 7. This extensive widespread digestion of the mesentery and omentum is not seen following the escape of the usual gastric or duodenal content. The occurrence of a chronic progressive ulcer in the isolated accessory stomach and the complete absence of any comparable lesion in the small channel left along the lesser curvature, the gastric canal or *magenstrasse*, is very instructive. The mucous membrane of this literal gastric canal has withstood the mechanical effect of swallowed food and the motility of gastric digestion, and in spite of this the long suture line has healed and remained so. In the accessory stomach, on the other hand, no food has entered and the mechanical effect of coarse food particles massaged into the mucosa by digestion peristalsis has been entirely absent. Nevertheless, an ulcer formed, became chronic and progressive, and finally perforated. The most evident difference between the two portions

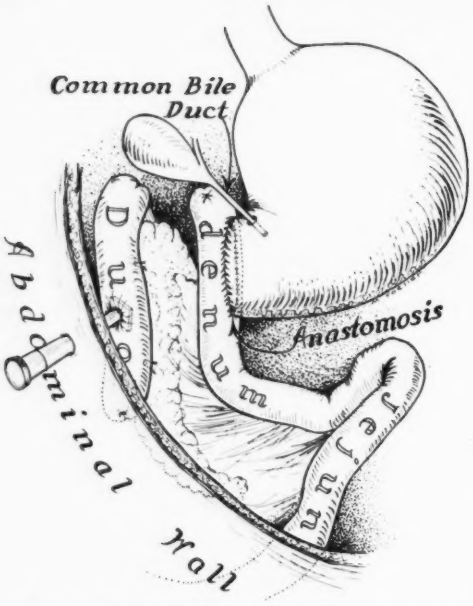

Fig. 16. Diagrammatic illustration of the type of pancreatic fistula used. The pancreatic ducts empty into the isolated closed segment of the upper duodenum and an external fistula has been made of this pancreatic duodenal pouch with a special gold plated cannula.

Fig. 17. Photograph showing a large chronic duodenal ulcer in a dog, discovered at autopsy 3 months after the production of a pancreatic fistula as illustrated in Figure 16.

of the stomach seems to lie in the probable acidity and pepsin concentration of their respective contents. In the one case, the isolated pouch, the free acidity and pepsin concentration was very high while in the other it is probable that its content would fall within the range of acidity and pepsin concentration of the stomach of the normal unoperated animal.

RELATION OF SWALLOWED SALIVA TO THE GENESIS OF GASTRIC ULCER

In the preceding section experimental evidence has been submitted indicating that the pure undiluted gastric juice may, by its chemical action, produce a chronic progressive ulcer in the mucosa of the ileum, jejunum, or stomach. We realize, of course, that only under the highly artificial conditions of these experiments does the mucosa of the alimentary tract become exposed to such pure gastric secretion, and it is our present purpose to inquire into the relative significance of the various factors which operate to reduce the acidity and pepsin concentration to that of the usual gastric content. In this connection it seems clear that the neutralizing effect of swallowed saliva plays at most a very minor rôle. Swanson (1917) demonstrated that removal of the salivary glands does not appreciably affect the volume or acidity of gastric secretion from a Pawlow pouch. Since he was interested primarily in the possibility of a hormone from the salivary glands affecting gastric secretion, he made no analyses of the acidity of the contents of the main stomach. No ulcers, however, were found in these animals. We are indebted to Dr. Mary Montgomery for our observations on 3 dogs provided with a total œsophageal fistula in the neck and a gastrostomy. All of the swallowed saliva, of course, escaped by the fistula and the animal was fed with an artificial pabulum administered by way of the opening in the stomach. These dogs were kept in good nutrition for about a year and suffered no ill effects from the loss of saliva. No evidence of ulcer or erosion was found in the stomach or duodenum on postmortem examination.

ABSENCE OF THE NEUTRALIZING EFFECT OF SWALLOWED FOOD IN THE GENESIS OF ULCER

A priori it would appear that the most important single factor in reducing the acidity

and pepsin concentration of the secretion of the gastric fundus should be the diluting and neutralizing effect of the food. Most of the proteins are capable of combining with hydrochloric acid and the products of pepsin hydrochloric acid digestion exert an inhibitory effect on the enzyme itself. It is quite probable that a part at least of the favorable result from the regimen of frequent feeding in the medical management of ulcer is due to this factor. The question is of definite practical importance because from time to time there appears in the literature a suggestion that ulcer patients be treated by duodenal tube feeding or the administration of food by means of a jejunostomy. The idea that such a method puts the stomach at rest is quite false so far as its secretory function is concerned. It has been repeatedly demonstrated in the experimental animal that the introduction of food in the duodenum or jejunum produces a copious secretion of gastric juice. The type of isolated stomach described by Dragstedt and Ellis was found to secrete as much as 2,800 cubic centimeters of gastric juice of high acidity in 24 hours when food passed directly from the œsophagus into the duodenum. Reasoning from this kind of evidence, duodenal tube feeding might be expected to permit the accumulation in the stomach of a gastric content of much higher free acidity than would occur when food is given by mouth, and such a method should be more apt to produce an ulcer in the stomach than to cure one. It is not practicable to feed a dog by duodenal tube, so recourse must be had to other types of experiments which reproduce the desired situation so far as possible.

Silbermann (1927) has reported the occurrence of ulcers in the stomach and duodenum of dogs subjected to repeated "sham feeding" experiments. A double œsophagostomy was done on 23 dogs and feeding was accomplished through the peripheral œsophageal opening. These dogs were allowed to eat from 40 to 60 minutes three times a day, the swallowed food escaping to the outside via the fistula. Ulcers developed in every case in from 14 to 49 days.

Buechner (1928) reported the occurrence of ulcers in the stomach of rats following the repeated injection of histamine. Buerkle-de la

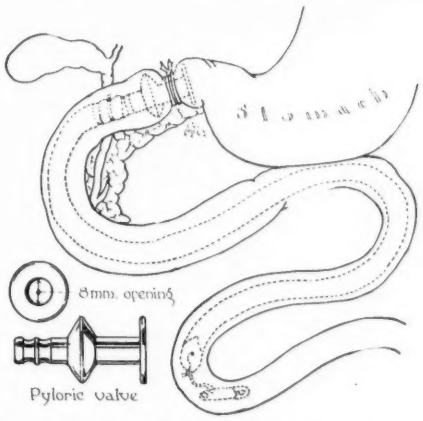

Fig. 18. Diagram illustrating the use of the valve to prevent regurgitation of duodenal juices into the stomach.

Camp (1929) found that the healing of acute ulcers produced by the injection of silver nitrate beneath the gastric mucosa of dogs was markedly delayed by the repeated injection of histamine. Both the ulcers occurring after sham feeding in dogs and those in rats injected with histamine have been attributed to the secretion of gastric juice in a stomach empty of food. The latter experiments are vitiated somewhat by the discovery of Hoelzel and Da Costa (1931) that ulcers may be produced in the pro-stomach of rats merely by protein restriction, a finding which emphasizes the necessity of adequate nutritional controls in all such work.

We have induced gastric secretion in the empty stomach by two methods but have not to date observed the production of ulcer in the previously intact gastric mucosa. The first observations were made on 3 adult dogs provided with a total œsophageal fistula and a gastrostomy. These animals were in excellent physical condition. When offered food they ate eagerly, but the partially masticated and ensalivated food escaped by way of the œsophageal fistula when swallowed, instead of passing into the stomach. Sham feeding of this type has been shown to produce a copious secretion of gastric juice as a result of reflex stimulation of secretory nerves reaching the stomach in the vagi. All 3 dogs were given such sham meals daily for a period of a month,

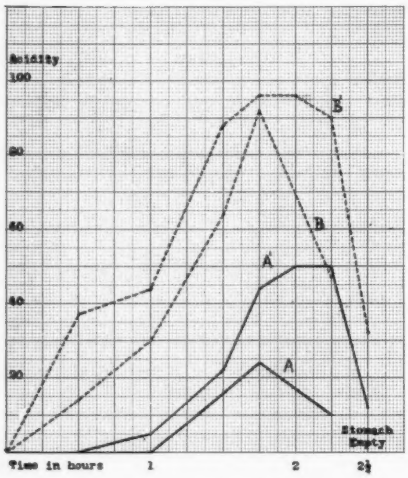

Fig. 19. Curves illustrating the effect of preventing duodenal regurgitation on the acidity of the gastric content after a test meal of meat and water. The uninterrupted lines (A and A′) represent free acidity, the broken lines (B and B′) total acidity. A and B are pre-operative, A′ and B′ postoperative curves. Dog 2.

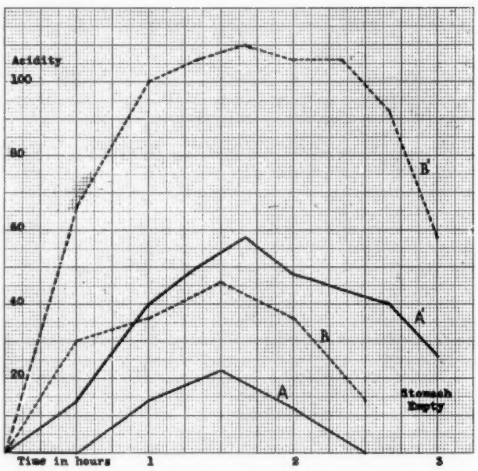

Fig. 20. Curves illustrating the effect of preventing duodenal regurgitation on the acidity of the gastric content after a test meal of meat and water. The uninterrupted lines (A and A′) represent free acidity, the broken lines (B and B′) total acidity. A and B are pre-operative, A′ and B′ postoperative curves. Dog. 3.

at the end of which time they were sacrificed and the stomach and duodenum carefully examined. No ulcers were found. These experiments, while decidedly limited in number, suggest that nervous or "appetite" gastric juice does not readily produce a lesion in the empty stomach.

We appreciate of course that this nervous stimulation of gastric secretion is only one phase of the normal mechanism and that perhaps the larger share of the total volume of juice produced is due to the stimulating effect of gastrin bodies elaborated as a result of the presence of food in the stomach or upper intestine. In the following preparation (Fig. 8) we have attempted to provide for the secretion of gastric juice in the empty stomach with both nervous and "humoral" stimulation and with the normal connections with the duodenum. The stomach was cut across at the cardia, care being used not to injure the vagus nerves supplying the stomach. The upper end of the stomach was infolded and closed. The jejunum was then cut across about 12 inches distal to the duodenojejunal junction, and the lower end of the divided jejunum brought up and anastomosed to the œsophagus. The upper end of the divided jejunum was implanted into the intestine by end-to-side anastomosis about 12 inches from the union with the œsophagus. Such a preparation should provide for a maximum secretion of gastric juice into the stomach empty of food. The presence of food in the mouth with its mastication produces a reflex stimulation of the vagi resulting in the so called appetite secretion and the subsequent entrance of the swallowed food into the upper jejunum provides for the elaboration of gastric secretin and the chemical stimulation of the gastric glands. No food should enter the stomach except as it might overcome the direction of peristalsis in the jejunum. The only factors remaining to reduce the acid pepsin concentration of the fundus secretion are the possible auto-neutralizing effect of gastric mucus or base secreted by the gastric mucosa and the regurgitation of alkaline secretions from the upper duodenum. We have no direct evidence that either of these factors were actually operative. Only one dog has been studied by this method to date (for 70 days) but in this animal no lesion developed in the stomach, duodenum, or jejunum. Obviously

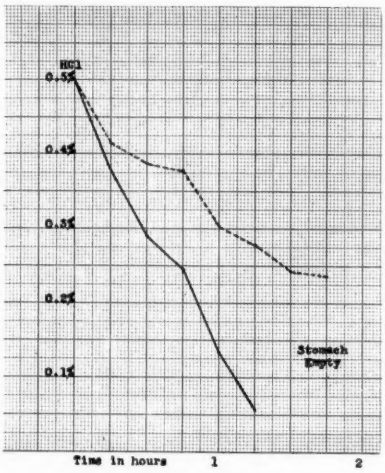

Fig. 21. Curves showing the neutralization of 200 cubic centimeters 0.5 per cent hydrochloric acid placed into the empty stomach before and after the introduction of the pyloric valve to prevent duodenal regurgitation. The broken line represents the postoperative results. Bile was present in the pre-operative aspirations consistently after the first ½ to 1 hour, but never after the valve was put in. Dog. 12.

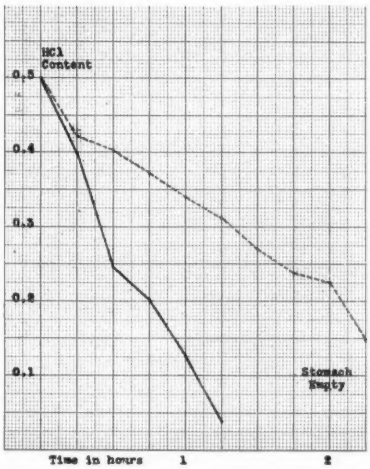

Fig. 22. Curves showing the neutralization of 200 cubic centimeters 0.5 per cent hydrochloric acid placed into the empty stomach before and after the introduction of the pyloric valve to prevent duodenal regurgitation. The broken line represents the postoperative results. Bile was present in the pre-operative aspirations consistently after the first ½ to 1 hour, but never after the valve was put in. Dog. 13.

more data are necessary before any final conclusions can be drawn, but the absence of an ulcer in this experiment is in harmony with the evidence detailed in the next section which indicates the great importance of the neutralizing effect of the bile and pancreatic juice.

RELATION OF THE NEUTRALIZING EFFECT OF THE DUODENAL SECRETIONS TO THE GENESIS OF ULCER

Exalto, in 1911, reported that he tied off the pylorus in dogs, performed a gastrojejunostomy, and then drained the duodenal juices of the proximal loop into the cæcum. Jejunal ulcers formed in 6 of 10 animals operated upon in this manner. He discussed his results in the light of the relatively high percentage of jejunal ulcers following the Roux type of gastrojejunostomy, which was popular in part of Europe at that time. The surgical duodenal drainage experiments of Mann and his associates, Williamson, Morton, and McCann, have been especially fruitful in demonstrating the significance of the neutralizing effect of the duodenal secretions in the cause of ulcer. This work has been widely confirmed in this country and later by Weiss and Hubster (1930) in Europe. The experiments discussed below afford further evidence concerning the significance of this neutralization of gastric chyme in the duodenum and also regarding the possible regurgitation of duodenal secretions into the stomach.

In one of the early procedures employed by Mann and his associates (1923) in their study of ulcer, the pylorus was cut across and the duodenal end infolded and closed. The jejunum was then divided just distal to the duodenojejunal flexure, the lower end united to the pyloric end of the stomach by end-to-end suture, and the proximal jejunum implanted into the ileum a short distance above the ileocæcal valve. A very high percentage of animals operated upon in this way developed chronic progressive ulcers in the jejunum a short distance from the line of anastomosis with the stomach. We have confirmed this observation. In repeating the experiment, however, we were impressed by the narrow lumen at the anastomosis and the great thickness of the musculature of the pyloric antrum. Both factors obviously might

Fig. 23. Photograph showing an acute gastric ulcer in a control animal 72 hours after the injection of 1.5 cubic centimeters of 5 per cent silver nitrate.

operate in a mechanical way to produce a mucosal lesion and prevent its healing. Mann himself recognized this point and believed that the motor drive of the stomach and the direction of the stream of gastric chyme were important in determining the site of the resulting ulcer. The following modification of Mann's experiment was devised to test the importance of these factors. Five dogs were operated upon as described except that the muscular pyloric antrum was first resected and a wide anastomosis made between the large open end of the stomach and the side of the jejunum (Fig. 9). A gastric outlet of this nature should not permit a narrow stream of acid chyme to be forcibly ejected against a single area of jejunal mucosa. However, in every case (100 per cent) jejunal ulcers developed (see Figs. 10 and 11). These ulcers were small and shallow and located at the lower end of the gastrojejunostomy, i.e., opposite the greater curvature of the stomach. They were not so deep or extensive as the ulcers following the end-to-end gastrojejunostomy, possibly because the "motor drive" effect was dissipated. These experiments were completed before the similar work of Ivy and Fauley (1931) was published and confirm their findings. These investigators reported that of 11 dogs operated upon in this manner, 5 developed jejunal ulcers. We may probably correctly infer then that the jejunal ulcers developing in this type of experiment are not dependent upon the "nozzle-like" effect of a narrowed pyloric orifice. We cannot, however, conclude from these experiments that the removal of a local neutralizing solution is the sole pathogenic factor in the ulcer formation. Both the animals operated upon by the exact technique of Mann and by our modified method developed a rapid and progressive cachexia. Immediately after the operation, all the dogs began to display a profuse watery diarrhœa which improved somewhat after a few days. The diarrhœa was usually accompanied by some loss of appetite. The weight loss was marked and progressive, and many animals lost 50 per cent of their original body weight in 1 to 2 months. They all died within 2 months in spite of freedom from laboratory infections. It seems likely that death was caused by the nutritional disturbance rather than the jejunal ulcers, most of which did not perforate nor cause excessive hæmorrhage. The factors in this nutritional disturbance are probably two-fold. The absence of two very important digestive secretions from the upper absorptive small intestine might be expected to interfere markedly with the digestion and absorption of food; and, second, the partial failure of reabsorption of pancreatic juice and bile, because of the low implantation, might be expected to result in dehydration and excessive loss of electrolytes from the blood and body fluids (Elman and McCaughan, Dragstedt, Montgomery, Matthews and Ellis). It is well recognized that cachexia produced by repeated hæmorrhage or infections (Ivy, 1920) or food deficiencies (Hoelzel, etc.) may operate to delay the healing of gastric lesions or induce ulcers. The cachexia is controlled by drainage of the duodenum higher into the jejunum, but under these conditions ulcers do not develop in the jejunum or the stomach. This failure of ulcer formation when the duodenum is implanted into the middle or upper jejunum has been attributed by Mann to a possible regurgitation of the duodenal secretions back orally through the jejunum and neutralization of the acid chyme in the region of the gastrojejunostomy. This interpretation seemed rather improbable to us and the following experiments were devised to test its validity. The results indicate that it is probably correct and in addition furnish further controls with regard to the significance of operative trauma

in the genesis of these "duodenal drainage jejunal ulcers."

Healthy adult dogs were secured and all operations were performed under complete ether anæsthesia and with the usual aseptic precautions. The procedure is illustrated in Figure 12. The pylorus was divided and the duodenum infolded and closed. The jejunum was then cut across just below the ligament of Treitz and the distal jejunum united to the pyloric end of the stomach by end-to-end suture. The distal end of the duodenum was implanted into the jejunum about 40 centimeters from the anastomosis with the stomach. No clamps were used in the operation. No. 00 chromic catgut was used for suture material and great care was taken not to injure the blood supply to the region of anastomosis. The animals promptly recovered from this somewhat extensive operation and remained in good condition thereafter. After a period of from 80 to 300 days, a second laparotomy was done and the region of the anastomosis between the jejunum and stomach carefully examined. In only 1 of the 21 dogs operated upon in this manner was a jejunal ulcer found, a striking contrast to the high incidence of such ulcers when the duodenal juices were drained into the lower ileum. To determine if this freedom from ulcer formation was due to the regurgitation of alkaline pancreatic juice and bile to the region of the gastrojejunostomy, a specially devised valve was then introduced into the segment of jejunum between the two anastomoses (see Fig. 12). This valve was so constructed as to permit of the flow of intestinal content only in one direction and so effectually prevented any regurgitation. The valve consisted essentially of an aluminum ring to which was attached a long piece (40 centimeters) of easily collapsible rubber tubing (Penrose). The ring was held in place in the jejunum by means of two tapes encircling the bowel, one being placed around the ring and one just below it. The inside diameter of the ring varied from 0.9 to 1.2 centimeters which was found adequate to prevent obstruction in most instances. Following the introduction of the valve at this second operation, the diet was restricted to finely divided and liquid foods.

Fig. 24. Photograph showing a chronic gastric ulcer in Dog 6, 30 days after the injection of silver nitrate, where duodenal regurgitation has been prevented by a valve in the pylorus.

The postoperative course varied somewhat. Five of the animals died of peritonitis due to a transection of the jejunum because of too tight a ligature about the aluminum ring. In six the ligatures were too loose, and the valve was passed with the fæces. Ten of the animals retained the valves and of these 6 developed progressive ulcers in the jejunum. The data are summarized in Table II.

TABLE II.—INCIDENCE OF JEJUNAL ULCER AFTER THE OPERATION ILLUSTRATED IN FIGURE 12.

Dog No.	Time between first and second operations	No. days after second operation when death occurred	Autopsy findings
1	None*	61	Chronic ulcer 2 cm. in diameter. Small perforation
2	170 days	63	No ulcer
3	173 days	42	No ulcer; dog died of hæmorrhage from erosion by valve into intestinal wall
4	97 days	157	Chronic ulcer, 1.5 cm. in diameter and 0.3 cm. deep. Perforated
5	96 days	54	Chronic ulcer, 1 cm. x 0.6 cm. Perforated
6	88 days	12	Acute ulcer, 0.4 cm. in diameter. Perforated
7	87 days	65	Chronic ulcer, very large and deep. Base made of liver. Perforated
8	101 days	46	No ulcer. Animal died of obstruction of valve by hairball
9	77 days	16	No ulcer. Animal died of obstruction of valve by hairball
10	70 days	81	Chronic ulcer, 2 x 1 cm. Perforated

*In Dog 1 all the operative work was done in one stage.

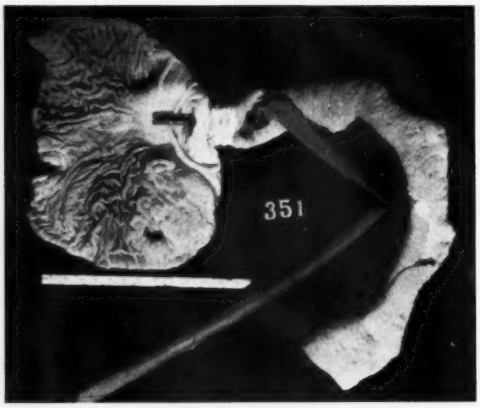

Fig. 25. Photograph showing a chronic ulcer in a trans plant of duodenum in the gastric wall where duodenal regurgitation has been prevented by a valve in the pylorus. The metal screw was placed inside the valve to indicate its position and the duodenum has been slit open to show the rubber tubing.

The photographs in Figures 13, 14, and 15 illustrate quite well the large size of these ulcers and their characteristic punched-out margins. It is significant that in each case the ulcer developed near the anastomosis with the stomach and at some distance from the valve. It is accordingly improbable that the valve exerted any local irritating effect of consequence in the genesis of the lesion. There was always a considerable inflammatory reaction around the valve. Numerous adhesions were present and the omentum was greatly thickened where it had been wrapped around the jejunum. The dogs remained in good physical condition following the second operation except those that died accidentally, as noted in Table II (i.e. Dogs 3, 8, 9). Each of the dogs (Dogs 2, 3, 8, and 9) which did not develop ulcer died from an accidental cause. It is possible that the incidence of ulcer formation might have been higher had it been possible to keep all the animals alive under the conditions of the experiment.

The data obtained in these experiments are instructive in that they rule out cachexia and surgical trauma as important factors in the genesis of the jejunal ulcer of the Mann experiment. The high implantation of the duodenal loop caused much less disturbance in digestion and absorption than when the bile and pancreatic juice passed into the lower ileum. The long interval between the first operation and the introduction of the valve permitted the anastomosis between the stomach and duodenum to become well healed and demonstrated that the trauma of operation and the mechanical factors of digestion operating were insufficient to produce a jejunal ulcer. The development of such an ulcer in a relatively high percentage of cases after the introduction of the valve, which so far as we can see acts only to prevent a regurgitation of the alkaline duodenal juices, is to our mind very strong evidence that this failure of neutralization of the acid gastric chyme is the factor of prime importance in such ulcer formation. The efficiency of the valves in preventing regurgitation of intestinal content is indicated by the following evidence. After the first operation, consisting of a gastrojejunostomy with high implantation of the duodenal loop, bile was frequently found in the gastric content but never after the introduction of the valve. Curiously enough, however, the acidity of the gastric content as determined by test meals was but little affected by the valve. At autopsy the jejunal mucosa above the valve was never bile-stained and water introduced into the intestine below the valve could not be forced by it into the upper jejunum.

OCCURRENCE OF CHRONIC PROGRESSIVE DUODENAL ULCER IN DOGS WITH PANCREATIC FISTULÆ

In 1930 Dragstedt, Montgomery, and Ellis described a new method for the construction of a permanent total pancreatic fistula in the dog. The upper portion of the duodenum, into which the various pancreatic ducts empty, was converted into a closed sac and connected to the exterior by means of a special gold plated cannula. The common bile duct was then implanted into the stomach and the continuity of the alimentary tract reestablished as indicated in the diagram in Figure 16. The dogs readily recover from this somewhat extensive operation, and if care is taken to replace the minerals lost in the pancreatic juice by the daily intravenous

administration of salt solution, they may be kept in fairly good nutrition in the laboratory for long periods. We have prepared a number of such animals in connection with other work. They all developed one, or more, very large chronic ulcers in the duodenum near the anastomosis with the stomach (Fig. 17). Several of these caused death from hæmorrhage or perforation. These observations confirm the findings of Elman (1931).

It is significant that we have been able to prevent this ulcer formation for periods as long as 6 months by the oral administration of calcium carbonate, sodium bicarbonate, or finely ground bone meal. In one such animal preserved in good nutrition for 5 months by this neutralization therapy, an acute ulcer developed in the duodenum and caused a fatal hæmorrhage 3 days after the therapy was stopped.

EFFECT OF THE PREVENTION OF DUODENAL REGURGITATION INTO THE STOMACH

The suggestion that the acidity of the fundus secretion of the stomach was under normal conditions partially neutralized by the regurgitation of duodenal secretions, was first elaborated by Boldyreff about 30 years ago. He claimed that such a regurgitation invariably occurred when the acidity of the stomach content approached that of pure gastric juice. For example, 200 cubic centimeters of 0.5 per cent hydrochloric acid placed in the empty stomach of the dog and removed 1 hour later was found to have an acidity of only 0.2 to 0.15 per cent and to contain both bile and pancreatic enzymes. If the pancreatic ducts were previously ligated or the pancreatic juice removed by means of a fistula, the neutralization proceded so slowly that little change was noted even after several hours. Pancreatic juice was considered more effective in this neutralization than the other duodenal secretions combined. Elman has recently confirmed and extended these findings of Boldyreff and has in addition reported the following highly significant observation. Three hundred cubic centimeters of 0.5 per cent hydrochloric acid introduced into the empty stomach of a patient suffering from an ulcer near the pylorus was much more slowly neutralized than in the normal human stomach. This he attributed to decreased duodenal regurgitation because of pylorospasm. A number of observers have noted the infrequent appearance of bile in the gastric content of patients with pyloric ulcer as compared with the normal.

The development of a valve similar to the one described in the preceding section which permits the passage of intestinal content in only one direction has made it possible for us to study the effect of preventing duodenal regurgitation on the acidity of the gastric content, on the healing of artificial wounds in the gastric mucosa, and on the production of ulcers in implants of intestinal mucosa in the gastric wall. For this work short tubes of gold-plated brass or aluminum were used, to one end of which a piece of thin walled collapsible rubber tubing (Penrose) about 40 centimeters in length was attached. The valve was placed in the pylorus as indicated in Figure 18, the rubber tubing extending into the lower duodenum. A heavy linen ligature placed about the pylorus was found sufficient to hold the valve in place. After some experimentation it was found that valves with an inside diameter of 8 millimeters were large enough to permit of normal emptying of the stomach.

Healthy adult dogs of average size (10 to 14 kilograms) were selected and all operations were performed under complete ether anæsthesia and with the usual aseptic precautions. An incision was made in the anterior wall of the stomach about 10 centimeters from the pylorus and the valve fixed in place as indicated above. At the same time an acute ulcer was produced on the posterior wall of the stomach by injecting 1.5 cubic centimeters of 5 per cent solution of silver nitrate just beneath the mucosa. The gastrotomy wound was closed with catgut. Intravenous fluids were given for 3 or 4 days after the operation and thereafter only liquids and finely divided food by mouth in order to prevent obstruction through possible clogging of the valve. Repeated gastric analyses were made both before and at varying periods after the operation.

a. *Effect of preventing duodenal regurgitation on the acidity of the gastric content.* For this determination a test meal similar to the

one described by McCann (1929) was employed. The animals were given no food for 24 hours before the examination and the stomach was then proved to be empty by lavage and aspiration. A meal of 80 grams of finely ground, raw, lean beef, and 250 cubic centimeters of water were given and fractions secured by aspiration every 20 to 30 minutes until the stomach was empty. The curves in Figures 19 and 20 taken from 2 typical experiments prove that the prevention of duodenal regurgitation has raised both the free and total acidity of the gastric content. It will be noted that not only are the free and total acidity higher after the operation than before, but the height of the acidity is sustained longer than before operation. Occasionally in a preoperative fractional analysis, one sample of gastric content in a series might show an unusually high free acidity. In several such cases the succeeding sample was slightly bile colored and of decidedly lower acidity. In these experiments before operation bile was usually not present except toward the end of the experiment. In no case was bile found in the stomach after the introduction of the valve.

b. *Effect of preventing duodenal regurgitation on the neutralization of acid introduced into the stomach.* In these experiments no food was given for 24 hours and the stomach proved to be empty by lavage and aspiration as before. Two hundred cubic centimeters of 0.5 per cent hydrochloric acid was then placed in the stomach and fractions removed every 15 minutes for examination until the stomach was empty. The curves in Figures 21 and 22 taken from two representative experiments indicate that the acid introduced into the stomach is rapidly neutralized in part at least in the normal animal but only much more slowly in the same animal after duodenal regurgitation has been prevented. These findings confirm the observations of Boldyreff many years ago.

c. *Effect of preventing duodenal regurgitation on the emptying time of the stomach.* The emptying time of the stomach was determined by a method similar to the one described by Ivy and Fauley (1929). Twenty-four hours after the last feeding the stomach was proved to be empty by lavage and aspiration. A meal consisting of 80 grams of ground lean beef, 40 grams of white bread, 60 grams of barium sulphate, and 300 cubic centimeters of water, mixed together to form a thick paste, was then given and eaten readily. Fluoroscopic examination was made immediately and thereafter every 30 minutes until the stomach was empty. The examinations were made with the dogs lying back down and held in position by a frame especially devised for this purpose. Both the gastric analyses and the emptying time tests were repeated many times on the same animal until the results became consistent. Several weeks were usually required for training. At first the animals were frightened and struggled somewhat but in a short time became gentle and even cooperative, apparently little disturbed by the experiments. During the early period 6 to 8 hours were frequently required before the stomach became empty whereas, after training, the same animal emptied the stomach quite regularly in 4 to 4½ hours. For 2 weeks after the introduction of the valve in the pylorus the emptying time was prolonged to 12 hours or more. After this time, however, the stomach seemed to become adapted to the presence of the foreign body and the emptying time ranged between 4½ and 5½ hours, or only slightly more than before operation. It is probable that the valves produced an actual slight pyloric stenosis, since the gastric peristalsis was noticeably more vigorous than before operation. The ulcers (see below) were not detected by fluoroscopic examination.

d. *Effect of preventing duodenal regurgitation on the healing of acute gastric ulcers.* After control observations had been made on the gastric response to a test meal and to the Boldyreff acid meal as already described, the animals in this series were operated upon and the special valve placed in the pylorus. At the same time an acute ulcer was produced on the posterior wall of the stomach by injecting 1.5 cubic centimeters of 5 per cent silver nitrate beneath the mucosa. Control animals were injected in a similar way with the same amount of silver nitrate. The injection produced an immediate local necrosis, the slough being digested away in about 48 hours, leaving a sharply circumscribed superficial

ulcer (Fig. 23). In the control animals these acute lesions invariably healed in 15 to 18 days, a confirmation of the observations of Friedman and Hamburger (1914) and L. R. Dragstedt (1917). In 13 animals in which duodenal regurgitation was prevented by the valve, healing of the acute lesion was delayed in 6 cases. The data are summarized in Table III. Three of the animals were sacrificed after 25, 26, and 32 days, respectively, and of these one ulcer was still present (25 days), the others being healed. Six animals were sacrificed after 30 days, and, in these, 3 ulcers were still present (Dogs 5, 6, and 9), the remainder being healed. Four were examined after 45 days. Of these 2 ulcers were still present and 2 were healed. Had it been practicable to examine these lesions at shorter intervals after operation, a more detailed account might be given of their rate of healing. All of the ulcers showed signs of healing except one, which was examined after 30 days and which seemed to have become chronic and progressive (Fig. 24). It had a thickened indurated base and the gastric content at postmortem examination was dark brown and contained blood. The remaining ulcers were shallow, not thickened, and a narrow rim of regenerating gastric mucosa could be seen around the border of each. Histologically, these healing ulcers did not present any signs of inflammation and the surrounding mucosa appeared normal.

TABLE III.—EFFECT OF PREVENTING REGURGITATION ON HEALING

Summary of the data indicating the effect of preventing duodenal regurgitation on the healing of acute lesions in the stomach

Dog No.	Length of experiment days	Condition of ulcer
1	32	Healed
2	26	Healed
3	25	Unhealed but healing; 1.4 x 1.0 cm.
4	30	Healed
5	30	Unhealed, healing; 1.0 cm. in diameter
6	30	Chronic ulcer; 1.5 cm. in diameter
7	30	Healed
8	30	Healed
9	30	Unhealed but healing; 0.8 cm. in diameter
10	45	Healed
11	45	Unhealed but healing; 0.4 cm. in diameter
12	45	Healed
13	45	Unhealed but healing; 0.4 cm. in diameter

e. *Effect of preventing duodenal regurgitation on the development of ulcers in intestinal transplants in the stomach.* The experiments of L. R. Dragstedt and Vaughn (1924), in which transplants of sections of intestine, of spleen, and kidney, into the stomach wall were found to be very little affected by the gastric content, have been referred to above. However, in view of the delayed healing of gastric wounds and the increase in gastric acidity when duodenal regurgitation is prevented as described in the preceding section, it seemed desirable to determine whether transplants of intestinal patches into the stomach would survive under the same conditions. Morton (1928) has attacked this problem in a slightly different manner but in general our results confirm his findings. He transplanted sections of intestine into the stomach of dogs otherwise normal and found that these persisted, the mucosa remaining unchanged. When he drained the duodenal juices into the lower ileum as in the Mann-Williamson technique, 3 of the 13 animals developed chronic ulcers in the transplants.

In our experiments transplants of intestinal mucosa into large gastric defects were made, as described by the above investigators. Great care was observed not to injure the blood supply to the transplant and only catgut was used for suture material. Sixteen dogs in all were operated upon, and in 4 transplants were taken from the duodenum, 4 from the jejunum, 4 from the ileum, and 4 from the colon. The operations produced little or no subsequent ill effects. However, several of the animals developed severe respiratory infections and it is interesting that of these 2 showed evidence of ulceration in the intestinal transplant (one a colon and the other a jejunal patch) when examined a month

TABLE IV.—EFFECT OF PREVENTING REGURGITATION IN TRANSPLANTS

Summary of the data with regard to the incidence of ulcers in intestinal transplants in the gastric wall where duodenal regurgitation has been prevented by a valve in the pylorus

Dog No.	Length of experiment days	Source of patch	Condition of patch
10	45	Duodenum	Two chronic ulcers 2.2 cm. diameter and 1.0 x 0.6 cm.
4	30	Duodenum	Two chronic ulcers 1 cm. diameter
11	45	Jejunum	No ulcer
5	30	Jejunum	No ulcer
12	45	Ileum	Large chronic ulcer 5 x 1 cm.
6	30	Ileum	No ulcer
13	45	Colon	No ulcer
7	30	Colon	Two chronic ulcers 3 x 5 cm. and 2 x 4 cm.

later. In all of the others the intestinal transplants were normal, examination being made through a gastrotomy opening at a second operation.

In 8 of the animals, which seemed in the best physical condition, a pyloric valve was introduced at the second operation, the remaining 8 being kept for control. They were then returned to their cages and the postoperative course carefully observed. It was decided to terminate the experiment short of 50 days since in our experience the pyloric valves can only rarely be made to remain in place for a longer period. Half of the animals were sacrificed 30, and half, 45 days after the second operation and postmortem examinations were made immediately. With the exception of the 2 animals already noted, both of whom had severe respiratory infections, the transplants in the control group persisted and the mucosa remained normal. The data with regard to the transplants in the stomach where duodenal regurgitation was prevented by the pyloric valve are summarized in Table IV. It will be noted that 4 of the 8 developed ulcers in the intestinal transplants and of these 2 occurred in the duodenal mucosa. One of these is illustrated in Figure 25.

SUMMARY

1. Experimental reproduction in the lower animal of a counterpart of the Meckel's diverticulum ulcer of man, by the implantation of a small isolated pouch of gastric wall into the jejunum and ileum, yielded chronic progressive ulcers in 85 per cent of trials in the former and in 100 per cent in the latter. This is a striking example of the susceptibility of an organism's living tissues to the irritant action of its own pure, active gastric juice.

2. A chronic progressive ulcer developed in a totally isolated stomach, in the entire absence of food or the motility of digestion, and where the important exciting factor was probably the high acid pepsin concentration of the pure gastric secretion.

3. A chronic progressive ulcer developed in the wall of a large Pawlow accessory stomach, which took no part in digestion, whereas the remainder of the stomach, consisting of a small tube made up of the lesser curvature (*magenstrasse* or gastric canal) and upon which the entire burden of gastric digestion depended, remained entirely normal.

4. The development of jejunal ulcers after surgical duodenal drainage as in the experiments of F. C. Mann and his associates is probably not due to mechanical factors or motility, since they occurred in 100 per cent of such animals where the muscular pyloric antrum was first resected and a very wide anastomosis made with the jejunum.

5. A repetition of the experiment of gastrojejunostomy and surgical duodenal drainage, as in the Mann experiment, but with implantation of the duodenum 40 centimeters below the anastomosis of the jejunum with the stomach, resulted in the development of only 1 ulcer in 21 experiments. This freedom of ulcer formation was due to the regurgitation of duodenal juices to the region of anastomosis, since the introduction of a valve to prevent such regurgitation led to the formation of chronic progressive jejunal ulcers in 6 cases.

6. Preventing the regurgitation of alkaline duodenal juices into the stomach of normal dogs, by fixing a valve in the pylorus, raised both the free and total acidity of the gastric content after a standard test meal, delayed the neutralization of 0.5 per cent hydrochloric

acid placed in the stomach, delayed the healing of acute ulcers in the gastric mucosa produced by the injection of silver nitrate, and caused the appearance of spontaneous ulcers in transplants of intestinal mucosa sutured into defects in the stomach wall.

7. The experimental evidence presented in this paper has been interpreted by the authors to afford substantial support to the view that the chemical action of pepsin hydrochloric acid (of the concentration found in pure gastric juice) can by itself alone produce a typical chronic progressive ulcer in the stomach, duodenum, jejunum, ileum, or colon. The resistance of these organs to the digestive action of pure gastric juice decreases progressively from the stomach to the colon. In the application of these findings to the problem of spontaneous ulcer in man, it should be emphasized that no evidence has been offered which contradicts the possible deterrent action of gastric motility and the mechanical action of coarse food in the healing of acute lesions of the stomach or duodenum. Of these three factors operative in healthy individuals, however, the chemical action of the gastric secretion seems to be the most important both in the production of the acute lesion and in its subsequent chronicity.

LITERATURE

ASCHNER, PAUL W., and KARELITZ, S. Peptic ulcer of Meckel's diverticulum and ileum. Ann. Surg., 1930, xci, 573.

BOLTON, CHARLES. Ulcer of the Stomach. p. 386. London: Edward Arnold, 1913.

BOLDYREFF, W. The self-regulation of the acidity of the gastric juice. Quart. J. Exper. Physiol., 1915, viii, 1.

BUECHNER, FRANZ. Magensaft, Gastritis, und peptische Geschwuere. Klin. Wchnschr., 1930, ix, 1.

BUECHNER, FRANZ, SIEBERT, P., and MOLLOY, P. J. Ueber experimentelle erzeugte akute peptische Geschwuere des Rattenvormagens. Beitr. z. path. Anat., 1928–1929, lxxxi, 391.

BUERKLE-DE LA CAMP, H. Zur Pathologie und Chirurgie der Peptischen Schaedigungen des Magen-Darmkanals. Deutsche Ztschr. f. Chir., 1929, ccxx, 31.

CARLSON, A. J. The secretion of gastric juice in health and disease. Physiol. Rev., 1923, iii, 1.

DRAGSTEDT, LESTER R. Contributions to the physiology of the stomach. XXXVIII. Gastric juice in duodenal and gastric ulcers. J. Am. M. Ass., 1917, lxviii, 330.

Idem. Failure of reabsorption of gastric and pancreatic juice as a pathogenic factor in some gastrointestinal disturbances. Am. J. Surg., 1931, xi, 544.

DRAGSTEDT, LESTER R., and ELLIS, J. C. The fatal effect of the total loss of gastric juice. Am. J. Physiol., 1930, xciii, 407.

DRAGSTEDT, LESTER R., MONTGOMERY, M. L., and ELLIS, J. C. A new type of pancreatic fistula. Proc. Soc. Exper. Biol. & Med., 1930, xxviii, 109.

DRAGSTEDT, LESTER R., MONTGOMERY, M. L., ELLIS, J. C., and MATTHEWS, W. B. The pathogenesis of acute dilatation of the stomach. Surg., Gynec. & Obst., 1931, lii, 1075.

DRAGSTEDT, LESTER R., and VAUGHN, A. M. Gastric ulcer studies. The resistance of various tissues to gastric digestion. Arch. Surg., 1924, viii, 791.

ELMAN, R., and HARTMANN, A. F. Spontaneous peptic ulcers of duodenum after continued loss of total pancreatic juice. Arch. Surg., 1931, xxiii, 1030.

EXALTO, J. Ulcus jejuni nach Gastroenterostomie. Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1911, xxiii, 13.

FRÉMONT. A note on the experimentally isolated stomach. Bull. Acad. de méd., 1895, xxxiv, 509.

FRIEDMAN, J. C., and HAMBURGER, W. W. Experimental chronic gastric ulcer; a second contribution to the experimental pathology of the stomach. J. Am. M. Ass., 1914, lxiii, 380.

HOELZEL and DA COSTA. Report read before the Society for Experimental Biology and Medicine, 1931.

IVY, A. C. Studies on the experimental gastric and duodenal ulcer. Am. J. Physiol., 1919–1920, xlix, 143.

Idem. Contributions to the physiology of the stomach. LII. Studies on gastric ulcer. Arch. Int. Med., 1920, xxv, 6.

Idem. Studies on gastric and duodenal ulcer. J. Am. M. Ass., 1920, lxxv, 1540.

IVY, A. C., and FARRELL, J. I. Contributions to the physiology of gastric secretion; proof of humoral mechanism, new procedure for study of gastric physiology. Am. J. Physiol., 1925, lxxiv, 639.

IVY, A. C., and FAULEY, G. B. The effect of hunger on the emptying time of the stomach. Am. J. Physiol., 1929, xci, 206.

Idem. Factors concerned in determining the chronicity of ulcers in the stomach and the upper intestine; susceptibility of jejunum to ulcer formation; effect of diet on healing of acute gastric ulcers. Am. J. Surg., 1931, xi, 531.

IVY, A. C., MORGAN, J. E., FARRELL, J. I. The effects of total gastrectomy. Surg., Gynec. & Obst., 1931, liii, 611.

LIM, R. K. S., IVY, A. C., and MCCARTHY, J. E. Contributions to the physiology of gastric secretion; attempt to prove that humoral mechanism is concerned in gastric secretion by blood transfusion and cross-circulation. Am. J. Physiol., 1925, lxxiv, 616.

LINDAU, ARVID, and WULFF, HELGE. The peptic genesis of gastric and duodenal ulcer; especially in the light of ulcers in Meckel's diverticulum and the postoperative ulcers in the jejunum. Surg., Gynec. & Obst., 1931, liii, 621.

MANN, F. C. Experimentally produced peptic ulcer. Surg. Clin. N. America, 1925, v, 753.

MANN, F. C., and WILLIAMSON, C. S. The experimental production of peptic ulcer. Ann. Surg., 1923, lxxvii, 409.

MATTHEWS, WARREN B. Production of intestinal ulcers by active gastric juice. Proc. Soc. Exper. Biol. & Med., 1931, xxviii, 960.

MCCANN, JAMES C. Experimental peptic ulcer. Arch. Surg., 1929, xix, 600.

Idem. Studies on the control of the acidity of the gastric juice. Am. J. Physiol., 1929, lxxxix, 483.

MORTON, C. B. Observations on peptic ulcer. I. A method of producing chronic gastric ulcer. A consideration of etiology. Ann. Surg., 1927, lxxxv, 207.

Idem. Observations on peptic ulcer. IV. Patch transplants of jejunum in the stomach. Ann. Surg., 1927, lxxv, 879.

SHAPIRO, P. F., and IVY, A. C. Gastric ulcer. IV. The experimental production of ulcer of the stomach by means of local anaphylaxis. Arch. Int. Medicine, 1926, xxxviii, 237.

SILBERMANN, I. S. Experimentelle Magen-duodenal-ulcuserzeugung durch Scheinfuettern nach Pawlow. Zentralbl. f. Chir., 1927, liv, 2385.

SWANSON, A. M. Removal of salivary glands and gastric secretion. Am. J. Physiol., 1917, xliii, 205.

WEISS, A. G., and HUBSTER, C. Sur la pathogenie des ulceres gastroduodenaux. Arch. franço-belges de chir., 1930, xxxii, 282.

WOLFER, J. A. Chronic ulcer of the stomach. Ann. Surg. 1926, lxxxiv, 89.

HYSTERECTOMY AND THE ARTIFICIAL MENOPAUSE

REVIEW OF LITERATURE, REPORT OF NINETY-ONE CASES

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THE vasomotor disturbances associated with the physiological menopause are currently believed to result chiefly from a derangement of the internal secretory mechanism of the ovary. This belief is based partly on the observation that such symptoms appear when the ovaries are removed prior to the time when the physiological menopause might be expected to take place. Certain clinical observations lead to a further conclusion, namely: that the time at which the menopause starts is dependent upon whether the uterus has been removed or not. This belief rests on the observation that menopausal symptoms appear earlier in women whose uteri have been removed than in those who retain this organ, even though in both groups one or both ovaries have been conserved. Whether the inciter of this premature menopause is solely the loss of the uterus, or results from injury to the blood supply of the ovaries while removing the uterus, or to some other set of circumstances, cannot be stated at present. Observations of many surgeons, however, indicate that the menopause appears prematurely following hysterectomy, no matter what its cause may be.

The material which serves as a basis for this conclusion is open to two criticisms: First, many observers have presented too few facts to substantiate their opinions. Second, many of the women operated upon as reported in the literature, have been too near the age when the physiological menopause should occur to make them valuable as material from which to draw such conclusions. In many cases, their symptoms were probably physiological in origin rather than pathological.

The present study was undertaken in order to collect evidence regarding the part played by hysterectomy in bringing on an artificial menopause. The literature was searched for reports of patients upon whom a hysterectomy had been performed some years before the physiological menopause would be expected. A follow-up study of patients operated upon at the John G. Clark Clinic of the Hospital of the University of Pennsylvania was also carried out.

Physiological menopause. Information was sought concerning the age at which the physiological menopause occurs in women not operated upon. This was done with the idea that the figures obtained would serve as a control to compare with the postoperative findings, and at the same time, might influence the selection of the age period for the present study. Sanes, in a study of 476 private patients not operated upon, found the average age at the time of the physiological menopause was 47.1 years. Of this group, only 5.2 per cent experienced symptoms before the age of forty. Norris, from a study of 200 women, found the average age of the menopause to be 48.9 years for married women, 47.7 years for widows, and 46.9 years for spinsters. Of his group, only 4 per cent experienced menopausal symptoms below the age of 41. Novak, in a study of 100 women, found that 3 per cent ceased to menstruate before the age of 40. Kish, after a

study of 455 women, found the menopause to occur in 10.5 per cent before the age of 40. The figures enumerated, based on 1,322 women not operated upon (Table I), show that only 5.6 per cent experienced menopausal symptoms before the age of 40. On account of the low incidence of physiological menopause for this period of life, 40 years of age was decided upon as the upper limit for the follow-up period.

CASE REPORTS FROM LITERATURE

In reviewing the literature, only those operative cases were selected in which the hysterectomy was performed before the age of 36 years, and in which at least one ovary was retained that had never been subjected to irradiation. Of some five thousand patients operated upon, and reported in the literature, the records of only 107 (Table I) (reference marked with asterisks) satisfied the requirements for the present study and gave sufficient clinical data to permit their use as the basis for an opinion, concerning the relationship between hysterectomy and the artificial menopause. Of these 107 patients, 53.2 per cent (57 patients) showed menopausal symptoms before the age of 40, whereas less than 6 per cent of women not operated upon suffered before this age (Table I). Of the 57 patients, information was available concerning the time of onset in 41 (Table II). This varied from immediately after operation to 7 years later, the average interval being 15.7 months. Thirty-three (80.0 per cent) of the 41 patients experienced their first symptoms within 2 years of operation, 38 (92.6 per cent) within 3 years.

PERSONAL OBSERVATIONS

Our own observations were made on patients who were operated upon in the John G. Clark Clinic of the Hospital of the University of Pennsylvania. These patients were selected on the following basis: (1) that hysterectomy had been performed before the age of 36; (2) that one or both ovaries had been conserved; (3) that at least one year had intervened between operation and the time of the last report; and (4) that no patient had received pelvic radium or roentgen therapy. These patients were followed either by mail or were seen in the follow-up clinic; in many instances information was obtained from both sources. Information was secured concerning the development of menopausal symptoms and concerning the menses, if they persisted following operation. Knowledge was sought of only one symptom of the menopause, namely "hot flashes." It was believed information pertaining to this one symptom would suffice, and at the same time would be easily obtained.

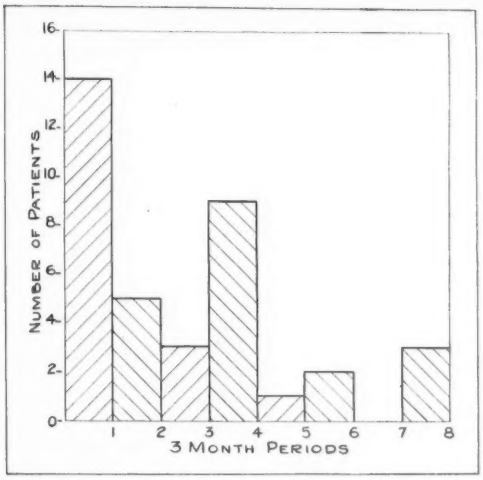

Fig. 1. Showing graphically the time interval between hysterectomy (before 36 years of age) and onset of "hot flashes" in a series of 37 women (Table II) exhibiting their first symptoms within 2 years of operation and before 40 years of age. The base line records time in 3 month periods; the vertical line the number of patients. Note the number of the latter (37.8 per cent) exhibiting first symptoms within 3 months of operation.

Information obtained concerning data were secured from 91 patients. Of these, 52 (57.1 per cent) were followed for 5 years or more, 57 (62.6 per cent) for 4 years or more, 69 (75.7 per cent) for 3 years or more, and 82 (90.1 per cent) for 2 years or more. The remaining 9 patients were followed from 1 to 2 years. The ages at the time of operation varied from 20 to 35 years, the average being 30.4 years.

The indications for hysterectomy in this series were equally divided between fibromyoma uteri and pelvic inflammatory disease.

Eighty-six of the 91 operations were of the supravaginal type; in the remaining 5 cases the entire organ was removed.

TABLE I.—INCIDENCE OF MENOPAUSAL SYMPTOMS UNDER 40 YEARS OF AGE

Condition	Source	Number of patients	Per cent with symptoms
No operation	Literature	1322	5.6
Hysterectomy	Literature	107	53.2
	Own	91	43.9

The incidence of menopausal symptoms in women under 40 years of age, not operated upon, and in those upon whom a hysterectomy with the conservation of one or both ovaries has been performed before the age of 36. Note that symptoms are 8 to 10 times more likely to occur following hysterectomy than when the uterus is not removed.

TABLE II.—TIME OF ONSET OF MENOPAUSAL SYMPTOMS FOLLOWING OPERATION

	Literature	Author's
Number of patients	41	40
Menopausal symptoms, percentage		
Within 2 years	80.0	92.5
Within 3 years	92.6	95
Menopausal symptoms—average time of onset in months	15.7	12.6*

The time of onset of menopausal symptoms before the age of 40 in women subjected to hysterectomy before 36 years of age. Note the large number in whom the symptoms were observed within two years of operation.

*This figure includes 3 patients experiencing first symptoms 3, 5, and 8 years respectively, after operation. Omitting these three cases, the average for remaining 37 is 8.5 months.

TABLE III.—RELATION OF MENOPAUSAL SYMPTOMS TO THE AMOUNT OF OVARIAN TISSUE RETAINED

	Ovaries retained			
	One	Per cent	Both	Per cent
Number of patients	54	100	37	100
Hot flashes				
Present	28	51.8	12	32.4
Absent	26	48.2	25	67.6

The influence of the amount of conserved ovarian tissue upon the incidence of menopausal symptoms, when one or both ovaries were conserved at the time hysterectomy was performed. Note the smaller number of patients having "hot flashes" in the cases where both ovaries were conserved instead of only one.

Of the 91 patients, 40 (43.9 per cent) experienced "hot flashes" before the age of 40 (Table I). Thirty-seven (92.5 per cent) of the 40 patients had their "hot flashes" within 2 years of operation (Figure 1 and Table II), and 31 (77.5 per cent) within the first year. Data concerning the intensity and duration of symptoms were too variable to be of value.

Thirty-one (36 per cent) of the 86 patients on whom a supravaginal hysterectomy was performed, menstruated following operation. Twenty were regular, 11 irregular. Only 10 (32.2 per cent) of these 31 menstruating patients had flashes, whereas 28 (50.9 per cent) of 55 non-menstruating patients had menopausal symptoms before the age of 40. From this comparison it is evident that the patient is less likely to have "hot flashes" if menstruation persists.

One ovary was removed from each of 54 patients. "Hot flashes" were experienced by 28 (51.8 per cent) of these women before the age of 40 years. In the 37 patients in whom both ovaries were retained, "hot flashes" were present in only 12 (32.4 per cent). A comparison of these figures indicates that the artificial menopause is less likely to occur when both ovaries are conserved than when one is removed.

The presence of the menses and the amount of ovarian tissue conserved (one or both ovaries) did not affect the time of onset or the intensity of the menopausal symptoms.

SUMMARY AND CONCLUSIONS

1. A series of 91 women on whom a hysterectomy was performed before the age of 36 years, with the retention of one or both ovaries, has been studied with reference to the incidence of menopausal symptoms occurring before the age of 40.

2. The frequency of these symptoms before the age of 40 was approximately eight times that occurring in a control group of women not operated upon.

3. Ninety-two per cent of the patients exhibiting menopausal symptoms before the age of 40, did so within 2 years of operation.

4. Thirty-six per cent of 86 women, on whom a supravaginal hysterectomy was performed, menstruated following operation.

5. Menopausal symptoms appearing before the age of 40 were noted more often in patients without menses than in the ones who retained them, in the ratio of 5 to 3.

6. Menopausal symptoms appearing before the age of 40 were observed more often where one ovary was removed than when both were retained, in a ratio of 5 to 3.

7. The time of onset of menopausal symptoms and their severity were approximately the same, whether one ovary was removed or both were left undisturbed.

8. From this study, it is concluded that hysterectomy hastens the onset of the menopause.

9. When a hysterectomy is necessary before the menopause, as much endometrium as possible should be retained to favor the continuation of menstruation, and both ovaries should be conserved, if possible.

BIBLIOGRAPHY

*1. ASCHNER, B. Ueber schaedliche Spaetfolgen nach Uterusexstirpation sowie operativer und radiotherapeutischer Kastration. Arch. f. Gynaek., 1925, cxxiv, 113–160.

*2. Idem. Untersuchungen ueber die Wirkung der Uterusexstirpation und der kuenstlichen Menopause. Wien. klin. Wchnschr., 1926, xxxix, 690–694.

*3. Idem. Ueber das spaetere Befinden von Frauen nach Uterusexstirpation mit Erhaltung der Ovarien. Zentralbl. f. Gynaek., 1929, liii, 1959–1965.

*4. BURCKHARD, G. Ueber die Dauererfolge der Myomoperationen. Ztschr. f. Geburtsh. u. Gynaek., 1900, xliii, 8–59.

*5. DORAN, A. H. G. Subtotal hysterectomy for fibroids: the after-histories of 60 cases: preservation of the ovary and the Abel-Zweifel theory. Lancet, 1905, ii, 1310–1318.

*6. ERDMANN, G. Folgezustaende der Uterusexstirpation bei Belassung von Ovarialsubstanz. Zentralbl. f. Gynaek., 1927, li, 3050–3060.

*7. JAYLE, F. Troubles de ménopause survenant 10 ans après une hystérectomie vaginale et à l'âge de la ménopause. Rev. franç. de gynéc. et d'obst., 1924, xix, 79–80.

*8. KEITLER, H. Ueber das anatomische und funktionelle Verhalten der belassenen Ovarien nach Exstirpation des Uterus. Monatschr. f. Geburtsh. u. Gynaek., 1904, xx, 686–753.

9. KISH. Quoted by Novak.

*10. KRAUL, L. Untersuchungen ueber die Wirkung der Uterusexstirpation und der kuenstlichen Menopause. Wien. klin. Wchnschr., 1926, xxxix, 297–300.

*11. LEOPOLD and EHRENFREUND, F. Ueber 151 vaginale Totalexstirpationen wegen Uterusmyomen und ueber den Einfluss der Erhaltung der Eierstoecke auf das spaetere Befinden der operierten. Beitr. z. Geburtsh. u. Gynaek. Rudolf Chrobak, 1903, ii, 135–165.

*12. MANDL, L., and BUERGER, O. Die biologische Bedeutung der Eierstoecke nach Entfernung der Gebaermutter. Leipzig and Vienna: F. Deuticke, 1904.

13. NORRIS, C. C. The menopause. Am. J. Obst., 1910, lxi, 203–215.

14. NOVAK, E. Menstruation and its Disorders. Gynecological and Obstetrical Monographs. pp. 126–127. New York: D. Appleton & Company, 1921.

*15. POLAK, J. O. A further study of the end-results of the conserved ovary. Am. J. Obst., 1918, lxxviii, 199–211.

16. SANES, K. I. The age of the menopause, a statistical study. Tr. Sect. Obst. Gynec. & Abdominal Surg., Am. M. Ass., 1918, 258–284.

*17. STEINHARDT, B. Ein Beitrag zur Frage der kuenstlichen Menopause. Ztschr. f. Geburtsh. u. Gynaek., 1927, xci, 361–385.

*18. THOMAS, G. C. The after histories of 100 cases of supravaginal hysterectomy for fibroids. Lancet, 1902, i, 294–300.

*19. WERTH, R. Untersuchungen ueber den Einfluss der Erhaltung des Eierstoeckes auf das spaetere Befinden der Operierten nach der supravaginalen Amputation und vaginalen Totalexstirpation des Uterus. Klin. Jahrb., 1902, ix, 529-602.

*Authors reported cases with sufficient data on which to base opinion.

EXPERIMENTAL STUDIES ON SUBARACHNOID ANÆSTHESIA

I. Paralysis of Vital Medullary Centers

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In spite of the really voluminous literature that has accumulated on the subject of subarachnoid anæsthesia, there still seem to be important questions on which authorities differ radically. This is so, perhaps, because most of the publications were written from the clinical standpoint or were reports of experiments performed with immediate clinical ends in view. The clinical phases of the subject, such as the preparation of the patient, the technique of injection, the advantages and disadvantages to the surgeon of this type of anæsthesia, have been the subject of exhaustive treatment. But the basic principles involved have not been sufficiently subjected to critical study. Such factors, for instance, as the possibility of paralysis of the medullary centers, the incidence of intercostal paralysis, the range and effects of the fall in blood pressure, and finally the etiology of complications and the measures indicated for combating them—for a knowledge of these fundamental questions, we have been dependent upon the opinions of authorities whose statements are too often based on clinical impressions rather than scientific investigation.

The effects of cocaine or its derivatives on the vital bulbar centers, i.e., the respiratory and vasomotor centers, seem to us to be the most important question involved.

Clinical authorities are divided into two camps on the subject of the paralysis of the vital medullary center. Dr. Labat, who is in charge of the section of Regional Anæsthesia of the Department of Surgery, New York University and Bellevue Hospital Medical College, states: "respiratory failure and all other symptoms associated with spinal anæsthesia are not due to the diffusion of the injected fluid to the brain and to the deleterious effects of the drug on the respiratory center," but rather to cerebral anæmia caused by the fall in blood pressure. In this he is seconded by Evans who states: "the respiratory depression is from bulbar anæmia, consequent upon the fall of blood pressure." Bryant states "that when death, due strictly to the anæsthesia method occurs in spinal anæsthesia cases, it is not due to toxic effect of an average dose of the anæsthetic drug nor is it due to paralysis by high diffusion of the latter, a common and erroneous belief. Death in such cases is due to cerebral anæmia." Babcock doubts "the existence of medullary paralysis from the proper use of spinal anæsthesia." Koster and Kasman by application of a pledget of cotton soaked in neocaine solution to the exposed closed portion of the medulla of frogs, guinea pigs, and cats, found that although the animals became anæsthetized, there was no effect on the respiration, and they concluded, therefore, that even in large concentrations procaine does not paralyze the respiratory centers.

On the other hand, Pauchet refers to "asphyxia by bulbar inhibition" due to ascension of the drug to the medulla and mentions having had three such cases in which artificial respiration was necessary. Pitkin believes that "it has been shown that the injection of tropacocaine, novocain, and stovaine into the spinal cord will cause immediate death with typical medullary symptoms." Sise attributed the fatality in 4 of 11 fatal cases he analyzed to "a dose of anæsthetic which rose in the spine and became placed very high, probably in the medulla." Rygh and Bessesen believed that the cause of 1 of their 2 deaths was the action of the drug on the respiratory center by its access to the fourth ventricle. Boyd and Yount attributed 2 of their 4 deaths to "spread of the drug to the medulla." Morrison speaks of "toxic impairment of the medullary center." And Allen states that "the immediate danger following spinal anæsthesia may occur as a result of paralysis of the respiratory, bulbar, vasomotor, and other higher centers."

[1]Read in part before the Clinical Congress of the American College of Surgeons, October 15, 1931

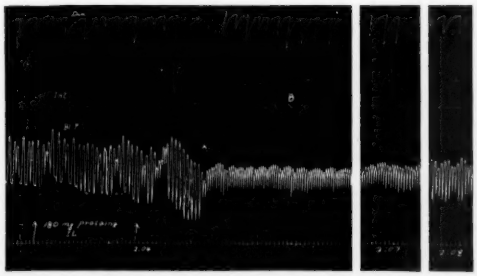

Fig. 1. Spinal injection. Dog 6—17.5 kilograms. *Dia.*, Diaphragmatic breathing; *Int.*, costal respiration; *Bl. P.*, blood pressure. At *A*, 15 seconds after injection, blood pressure fell slightly, the change in pulse pressure being more marked. At *B*, 30 seconds after the injection, the costal respiration starts to decrease in amplitude.

2:07 strip: costal respiration is almost completely paralyzed.

2:08 strip: costal respiration is completely paralyzed. Diaphragmatic contractions, however, keep on going. Both the systolic and pulse pressures are recovering.

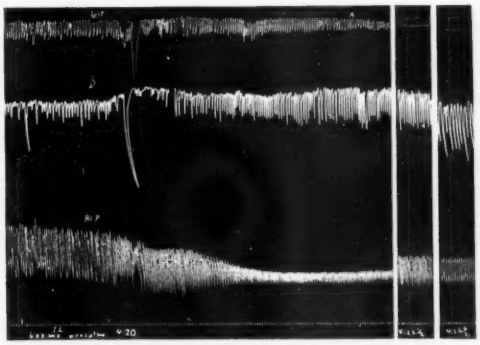

Fig. 2. Spinal injection. Dog 9—18 kilograms. *Int.*, Costal respiration; *D*, diaphragmatic respiration; *Bl. P.*, blood pressure. The blood pressure starts to fall before injection is complete. The fall in both systolic pressure and pulse pressure is more marked than in Figure 1. At *A*, the costal respiration starts to decrease in amplitude.

4:22½ strip: the costal respiration is almost paralyzed, the diaphragmatic respiration has increased in amplitude, the blood pressure is recovering in both systolic and pulse pressure.

4:26½ strip: the costal respiration is paralyzed and the diaphragmatic has increased further in amplitude.

Experimentally, most workers in this field agree that a paralysis of the medullary centers occurs when a large enough dose of cocaine derivative is made to reach the medulla. Aducco showed that the applications of crystals or of a salve of cocaine on the floor of the fourth ventricle of a dog led to respiratory paralysis. Jonnesco and Jiano obtained respiratory paralysis by injection of stovaine into the mid-cervical subarachnoid space and thought the paralysis to be central in origin. Bellelli also could get respiratory paralysis by injecting novocain through the atlanto-occipital membrane, and Janossy studying the action of coramine on the respiratory center, used the paralyzing effect of tropacocaine injected cisternally as a basis of experimentation. And recently Vehr, injecting procaine into the cisterna magna, came to the conclusion that only very large doses can cause respiratory paralysis. Jonnesco and Jiano, Bellelli, Janossy, and Vehr, since they show no tracings, leave uncertain the question whether the paralysis of the respiratory mechanism was central.

The most convincing work was done by Bloch, Camus and Hertz, in 1921, who demonstrated that stovaine and syncaine injected through the atlanto-occipital membrane of a dog caused a sequence of symptoms terminating in paralysis of the respiratory center; that by artificial respiration the animal could be made to live; and that caffeine injected intracisternally was of decided benefit. Camus, in 1922, with tracings, showed the same phenom-

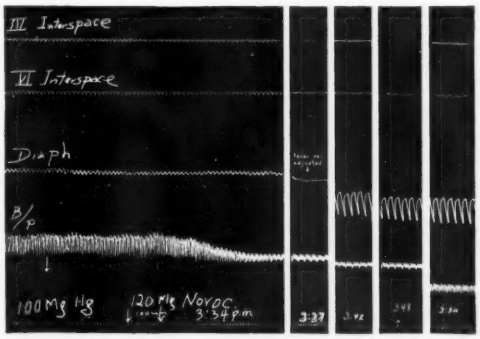

Fig. 3. Spinal injection. Dog 11—weight 20 kilograms. III, Costal respiration registered from third interspace; VI, same from sixth interspace; *Diaph.*, diaphragmatic respiration; *B/p*, blood pressure.

3:37 strip: the breathing from the sixth interspace is decreasing in amplitude, that from the III space is normal except for decreased rate. The diaphragmatic tracing is an artifact due to too tight a lever.

3:42 strip; VI is paralyzed, III is decreasing in amplitude, the apparent increase in diaphragmatic tracing is due to readjustment of the lever.

3:48 strip: III is paralyzed, VI registers negative waves due to sinking in of thorax during inspiration.

3:54 strip: both III and VI register negative waves. The diaphragmatic excursions show increased amplitude.

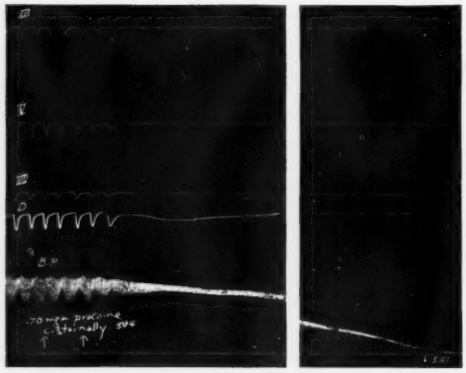

Fig. 4. Cisternal injection. Dog 7—17 kilograms. III, Respiration registered from third interspace; V, same from fifth interspace; VII, same from seventh interspace; *D*, diaphragmatic respiration; *B. P.*, blood pressure. In 10 seconds after injection, III, V, VII, and *D* are simultaneously paralyzed. The blood pressure gradually falls, reading 0 at 5:07, 3 minutes after injection.

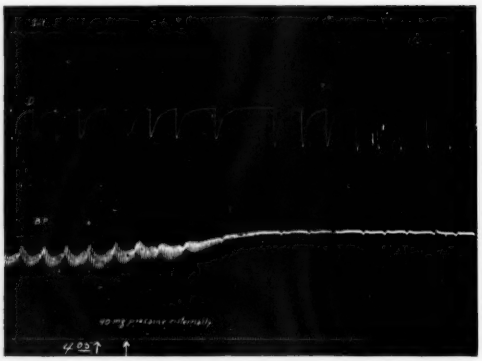

Fig. 5. Cisternal injection. Dog 6—15.5 kilograms. *Int.*, Costal respiration; *D*, diaphragmatic; *B. P.*, blood pressure. Note the prolonged rise of blood pressure outlasting the slight period of respiratory depression.

enon with novocain, and Soupalt, in 1923, could identify the same sequence of events in a patient who had 10 centigrams novocain injected into the spinal canal between the first and second lumbar interspaces. The work of the four mentioned authors has not had the recognition it merits.

In view of the divergence of opinion as detailed, it seems desirable to submit the subject to a reinvestigation. The following study was undertaken with the object of determining (1) the effects of procaine upon the respiratory and vasomotor centers, and (2) the efficacy of measures of resuscitation.

The work will be presented in the following order:

I. Injection of procaine into the spinal canal, the subject of a separate study, is introduced here as controls, to make clear our methods, reasoning and conclusions.

II. Injection of procaine into the cisterna magna as the best method of getting the drug in contact with the medullary centers in the intact animal.

III. Localization of the action of procaine injected cisternally.

IV. Resuscitation measures.

V. Difference in lethal dosages in normal and anæsthetized animals.

Dogs were used in these experiments. Each was given morphine hydrochloride 5 milligrams per kilogram subcutaneously and sodium amytal 25 milligrams per kilogram intravenously.

A pneumograph applied around the chest and connected to a tambour recorded the costal breathing, while a string attached to the anterior abdominal wall and made to pull on a lever recorded the diaphragmatic breathing. A brief explanation of the mechanism of these two systems of registration may be pertinent. During inspiration the chest expands, and the abdomen bulges. The expansion of the chest stretches the pneumograph and produces a partial vacuum in the closed system. The membrane of the tambour, therefore, falls and the lever falls with it. The bulging of the abdomen slackens the string attached to it, allowing the lever to fall of its own weight. During inspiration, therefore, both the costal and diaphragmatic levers fall, while during expiration they both rise. The blood pressure was recorded by connecting a registering mercury manometer with the femoral artery.

Spinal injections. When procaine is injected into the subarachnoid space at the first lumbar space in dosages of 100 to 600 milligrams the following events take place (Figs. 1 and 2). There is at first a fall in blood pressure and a fall in pulse pressure. The respiration, both costal and diaphragmatic, is at first quickened in response to the fall in blood pressure, then

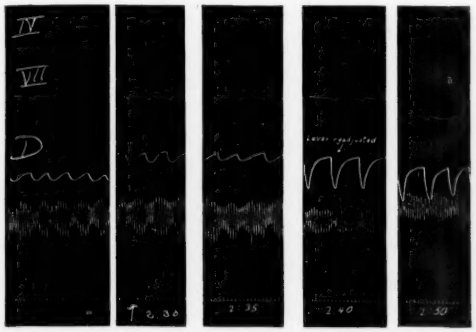

Fig. 6A. Localization experiment. Dog 13, weight 15 kilograms. Upper cervical laminectomy exposing closed portion of medulla and upper cervical cord. IV, Costal respiration registered from fourth interspace; VII, same from seventh interspace; *D*, diaphragmatic respiration. Cotton pledget soaked in 2½ per cent procaine solution applied to exposed closed portion of medulla at time marked by arrow in strip 2:30. The apparent increase of diaphragmatic excursions is due to readjustment of lever previous to the taking of the tracing. No intercostal or diaphragmatic paralysis occurred in the period from 2:30 to 2:50.

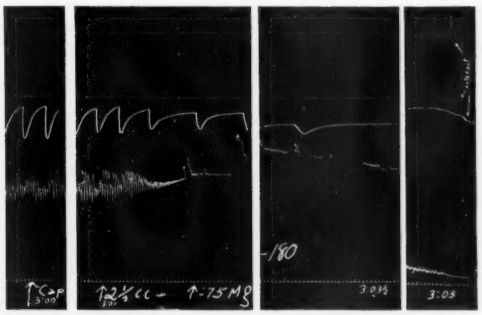

Fig. 6B. Continuation of localization experiment. No respiratory paralysis even at 3:00, 30 minutes after application of the drug. Capillary pipette introduced into fourth ventricle at time marked by first arrow.

3:01 strip: first part shows no apparent change in respiration or blood pressure caused by introduction of the pipette. Between second and third arrows in strip 3:01, 2½ cubic centimeters of the same solution was run through the pipette into the fourth ventricle. Note immediate decrease in respiratory rate and amplitude, and rise in blood pressure. The irregular line on the blood pressure curve is due to improperly writing blood pressure lever.

3:03½ strip: blood pressure is 180 millimeters, the respiration is paralyzed.

3:05 strip: the costal and diaphragmatic respirations both are paralyzed, the hump on the diaphragmatic curve is due to loss of tone of the diaphragm. Blood pressure has fallen to 0. The irregularities at the end of the respiration tracings are due to mechanical causes.

the rate is slowed; then dissociation occurs in that the amplitude of the costal breathing is gradually reduced, until at the end of some 3 to 18 minutes, depending upon the dose, the bulk of injected fluid and the speed of injection, it is entirely stopped. The diaphragmatic contractions on the other hand are not affected at this time and in fact, in Figures 2 and 3, they seem to compensate for the intercostal paralysis by increased amplitude. By the application of two or even three pneumographs at different levels of the chest above the eighth rib, it can be shown that this costal paralysis is ascending; that is, the intercostal nerves are paralyzed one after another as the drug diffuses upward (Fig. 3).[1] This intercostal paralysis is interpreted as peripheral; that is, paralysis of the nerve roots as they emerge from the cord.

Injection into the cisterna magna. When procaine is injected into the cisterna magna, however, an entirely different picture is presented (Fig. 4). As small a dose as 60 to 70 milligrams in a 17 to 20 kilogram dog produces a swift simultaneous stoppage of both intercostal and diaphragmatic respiration, and a fall in blood pressure without any preliminary rise in response to asphyxia, as would be expected from a normal vasomotor mechanism. Sometimes, however, when the dose injected is small, the blood pressure registers a rise (Fig. 5). The effects on the vasomotor mechanism will be taken up later.

The small size of the dose injected and the simultaneous cessation of the whole respiratory mechanism and fall in blood pressure, suggest an effect on the respiratory and vasomotor centers themselves. The fact that the cisterna magna communicates by the foramina of Luschka and Magendie with the fourth ventricle, on the floor of which are located the centers, makes the probability of a direct effect very strong.

Two other possibilities, however, must be considered. First, a paralysis of the phrenic and intercostal roots by a diffusion of the drug from the cisterna downward. Offhand, if this

[1] Pneumographs applied to the chest at a lower level than the eighth rib will not show purely costal breathing, especially when the drum is run at a slow speed and the tracings are too close together for analysis. The diaphragmatic digitations inserted from the eighth rib downward cause the ribs to move even if the intercostals are paralyzed.

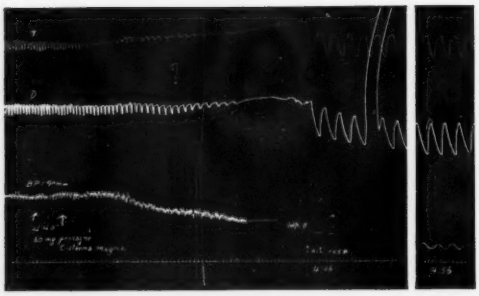

Fig. 7A. Resuscitation experiment. Dog 21, weight 20 kilograms. Cisternal injection. *T*, Costal respiration; *D*, diaphragmatic; *BP*, blood pressure. Note the respiratory paralysis 40 seconds after injection. Artificial respiration started at 4:46 marked by third arrow. The irregularities at *A* on the blood pressure curve are due to poorly writing lever.

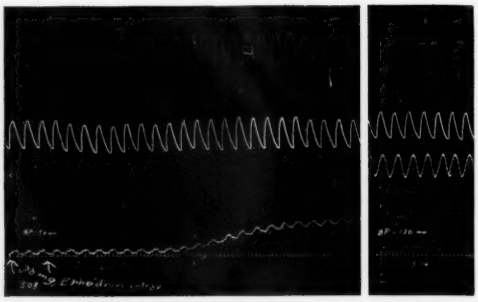

Fig. 7B. Resuscitation experiment continued. At 5:08, marked by the two arrows, 30 milligrams ephedrine hydrochloride is given intravenously. Note latent time in rise of blood pressure.

5:14 strip: blood pressure has risen to 130 millimeters.

were true, we would expect from analogy with our spinal experiments a paralysis of the diaphragm to take place first, followed by that of the intercostals in a descending order. This, however, did not occur. The second possibility is the permeation of the drug into the cord substance in sufficient quantity to paralyze the neurones, arising from the respiratory center and running in the cord down to the anterior horn cells of the phrenic and intercostal nerves. The localization experiment described below excludes the last two explanations.

Localization of action. An upper cervical laminectomy was done, the atlas, the second cervical vertebra, and a part of the basilar portion of the occipital bone being removed. This operation opened the cisterna, thus exposing the inferior aspect of the cerebellum, the closed portion of the medulla, and the upper cervical cord. A pledget of cotton soaked in 2½ per cent solution of procaine in cerebrospinal fluid, carrying about 5 cubic centimeters representing 150 milligrams of the drug was placed over the closed portion of the medulla and the upper cervical cord. The head of the dog was then tilted slightly upward to allow gravitation of the excess fluid downward. Tracings taken in the course of 30 minutes showed neither respiratory paralysis nor appreciable changes in the blood pressure (Fig. 6). This shows that whatever diffusion downward or whatever permeation of the drug into the cord had occurred this was not sufficient to paralyze either the phrenic and intercostal roots or the neurones going out of the center. Koster and Kasman in a similar experiment on a cat and guinea pig, obtained results differing from those here reported. The difference in the technique employed may serve to explain our diametrically opposite results.

A curved, blunt, capillary pipette was slowly introduced through the posterior medullary vellum into the fourth ventricle, care being taken not to injure the floor of the fourth ventricle by keeping close to the vermis of the cerebellum. Tracings taken for about a minute after the introduction of the pipette showed that no change due to the mechanical introduction had occurred either in the respiratory or blood pressure curves (Fig. 6A). Then 2.5 cubic centimeters of the solution containing 75 milligrams of procaine was allowed to flow by gravity through the pipette into the fourth ventricle. In less than 30 seconds the costal breathing stopped, followed soon after by paralysis of the diaphragm (Fig. 6B). The blood pressure registered a rise, then fell to zero, and the dog died.

Our explanation for the occurrence of paralysis when injection was made into the cisterna and for the non-occurrence of paralysis when application was made to the closed portion of the medulla, is that in the first case, in the closed system of an intact cisterna magna, diffusion could occur from the cisterna through the foramina to the fourth ventricle; while in the second case, the fluid having been spilled, no vehicle for diffusion was present. To verify

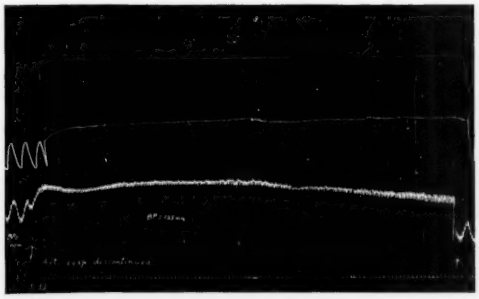

Fig. 7C. Resuscitation experiment continued. At 5:30 (point of arrow), artificial respiration discontinued. Note rise in blood pressure in response to asphyxia, showing recovery of vasomotor center. Note continued paralysis of respiration. Artificial respiration started again at time marked by second arrow.

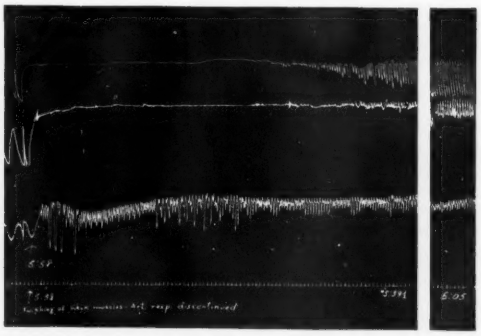

Fig. 7D. Resuscitation experiment continued. Twitching of neck muscles seen at 5:58—so artificial respiration is discontinued. Note the recovery of spontaneous respiration.

the correctness of this explanation, methylene blue was injected into the cisterna magna of an ntact dog in one case and applied to the closed portion of the medulla exposed by an upper cervical laminectomy similar to the one described above in another case. On examination of the brains at the close of the experiment, it was found that in the case of injection into the cisterna the fourth ventricle was colored blue, whereas in the animal in which the methylene blue was applied to the exposed medulla and upper cervical cord, the floor of the fourth ventricle showed no traces of methylene blue.

In the case of blood pressure, whether a rise or fall is registered depends upon the dose. In Figures 4 and 7A, there was a fall from the beginning of the experiment, in spite of the asphyxia occasioned by the respiratory cessation. This would indicate a primary paralysis of the vasomotor centers. In the experiments with smaller dosage (Figs. 5 and 6B), there may be at first a rise in blood pressure, then a gradual fall to zero in spite of the institution of artificial respiration or a primary rise without any subsequent fall. Whether this rise is due to the asphyxia, to stimulation of the vasomotor center, or to a combination of both factors or to other factors, yet unknown, we are unprepared to say. The fact that paralysis of the respiratory centers occurs first would suggest either that the vasomotor center is not anatomically as exposed to the procaine or that it is more resistant. The finding of Tatum and his co-workers that in poisoning by cocaine systematically administered, the respiration stops before the heart, is interesting in this connection.

Resuscitation experiment. With this evidence pointing to respiratory and vasomotor paralysis due to the injection of suitable doses of procaine into the fourth ventricle or into the intact cisterna magna, the subsequent procedures were carried out in an attempt to evaluate the efficacy of certain resuscitative measures. A 20 kilogram dog was given 60 milligrams of procaine hydrochloride at 4:45 marked on the tracing by the two arrows in Figure 7A. As will be seen from the graph, there was respiratory paralysis and a steady decline of the blood pressure, no asphyxial rise being in evidence. Artificial respiration was started 30 seconds after respiratory stoppage, but the blood pressure continued to fall to about 5 millimeters. At 5:08 (Fig. 7B), 23 minutes after the injection, 30 milligrams of ephedrine sulphate was injected into the femoral vein; after a latent time of 38 seconds, the blood pressure started to rise until at 5:14 it was above normal. At 5:33 (Fig. 7C) the artificial respiration was discontinued to determine if the respiratory center had recovered. Such interruptions of the artificial respiration were repeated at 5:43 and 5:53, but recovery had not yet taken place. At 5:58 (Fig. 7D), 1 hour and 13 minutes after the injection, twitchings of the neck muscles were seen. These twitchings were interpreted as the beginning of return of spontaneous activity of the respiratory

TABLE I.—NORMAL DOGS

Weight (kg.)	Non-lethal dose procaine (mg.) Cisternally
15	104
18.5	100
12	90
14	115
11.5	102
10	106
*7.5	90
* 8	105
* 5	90

*From Vehr's series.

TABLE II — FATAL DOSES

MORPHINE-SODIUM AMYTAL DOGS*

Weight (kg.)	Fatal dose procaine (mg.) Cisternally
7.5	50
20	60
17	70
18	70
11.75	60
19	65
13	30

SODIUM AMYTAL†

15.5 (40 mg. sodium amytal per kilo)	55
11.5 (45 mg. sodium amytal per kilo)	70

*Morphine hydrochloride 5 milligrams per kilogram was given subcutaneously and sodium amytal 25 milligrams per kilogram intravenously before the experiment.
†Given intravenously.

center. The artificial respiration was discontinued, and in little over a minute, both the intercostal and diaphragmatic breathing recovered spontaneously. At the end of the experiment, therefore, the dog was, to judge from the respiratory and blood pressure tracings, as well as it was when the experiment started.

Differences in the fatal intracisternal dose in anæsthetized and unanæsthetized dogs. Normal dogs require a much larger dose of procaine intracisternally to kill than dogs anæsthetized with morphine and sodium amytal or with sodium amytal alone. The two tables, one representing comparatively large doses that failed to kill normal dogs (Table I) and the other representing fatal doses in dogs anæsthetized with morphine and sodium amytal or with sodium amytal alone (Table II), will show the difference. Some of the experiments in Table I are from Vehr's series. This finding, that a barbituric acid derivative reduces the lethal dose intracisternally of procaine hydrochloride, is apparently at variance with the work of HofVendahl, Tatum et al, La Mendola, and Martin, who showed that members of the barbituric acid series raise the minimum lethal dose of cocaine administered subcutaneously. The discrepancy, however, is more apparent than real. In cocaine poisoning brought about by the systemic administration of cocaine, the convulsive phase is the striking feature, whereas in injection into the cisterna magna, the paralytic phase is predominant. The works of Jacobj and Roemer, Impens, Jackson, and Bouckaert showing that the barbitals in large doses have a depressing effect on respiration, explain the increased vulnerability to procaine of animals narcotized with sodium amytal.

This lowering of the lethal dose of procaine injected cisternally by the previous administration of barbituric acid derivatives is significant in view of the increasing tendency to use them as a "basal anæsthetic" in cases of spinal anæsthesia. According to our findings, it is distinctly contra-indicated, and is a misapplication of the findings of HofVendahl, Tatum, and others.

The work now in progress is an investigation of the effect of certain pathological conditions and of drugs other than sodium amytal on the resistance of the medullary centers to the paralyzing effect of procaine.

SUMMARY

1. Procaine through direct action on the medulla produces respiratory and vasomotor paralysis when injected in sufficient concentration into the cisterna magna.

2. When complete paralysis occurs, artificial respiration and the intravenous injection of ephedrine are effective as measures of resuscitation.

3. In sodium amytal and in combination morphine-sodium amytal narcosis, there is a lowered resistance of the centers to the paralyzing effects of procaine.

This study was made at the suggestion of Professor George David Stewart of the department of surgery and of Dr. Arthur M. Wright, director of the New York University Surgical Division of Bellevue Hospital, to both of whom we wish to acknowledge our thanks for their constant interest and encouragement throughout the course of this work. We also take pleasure in thanking Professor George B. Wallace of the department of pharmacology for his helpful suggestions and criticism.

BIBLIOGRAPHY

1. Aducco. Ann. Ped. e Sc. Aff., 1889.
2. Allen, C. W. Local and Regional Anesthesia. p. 472. New York: W. B. Saunders Company, 1918.
3. Babcock, W. W. Spinal anesthesia, an experience of 24 years. Am. J. Surg., 1928, v, 574.
4. Bellelli, F. Contributo Sperimentale all rachianestesia sottooccipitale. Folia med., 1928, xiv, 691.
5. Bloch, R., Camus, J., and Hertz. Rachistovainisation et Rachisyncainisation Expérimentales: Leurs Accidents, Les Moyens d'y Remedier. Compt. Rend. Soc. de biol., 1921, lxxxv, 297.
6. Bouckaert, J. Influence du somnifeine sur l'élimination carbonique, le volume Respiratoire et la température du Lapin. Arch. internat. de Pharmacol. et de Therap., 1926, xxxi, 357–366.
7. Boyd, A., and Yount, C. C. Routine spinal anesthesia. J. Am. M. Ass., 1917, lxviii, 601.
8. Bryant, J. M. Physiology of spinal anæsthesia. J. Florida M. Ass., 1930, xvi, 345–355.
9. Camus, J. Action de la stovaine et de la novocaine sur les centres bulbaires. Paris méd., 1922, xii, 205.
10. de Waele, H. L'action de doses élevées des dérivés barbituriques sur le pneumogastrique. Arch. internat. de Physiol., 1925, xxv, 83–87.
11. Evans, C. H. Spinal anesthesia—possible complications. Am. J. Surg., 1928, v, 581; Spinal Anesthesia. pp. 103–110. New York: Paul B. Hoeber, Inc., 1929.
12. Gruber, C. M., and Baskett, Roy F. The effect of phenobarbital (luminal) and sodium phenobarbital (luminal-sodium) upon blood pressure and respiration. J. Pharmacol. & Exper. Therap., 1925, xxv, 219.
13. HofVendahl, A. Die BeKaempjung der Cocainvergiftung im Tierversuch. Biochem. Ztschr., 1921, cxvii, 55–56.
    Ibid. Die BeKaempfung der Cocainvergiftung im Tierversuch. Ztschr. f. Hals. Nasen u. Ohrenheilk., 1922, i, 233–234.
14. Impens, E. Pharmak. Luminal oder Phenylethylbarbitursaeure. Deutsch. med. Wchnschr., 1912, xxxviii, 945–947.
15. Jackson, D. E. Action of isopropylethylbarbituric acid (ipral). J. Lab. & Clin. Med., 1922, viii, 23–32.
16. Jacobj, C., and Roemer, C. Untersuchungen zur Pharmakologie des Veronals: Einfluss auf Temperature, Atmung und Zirkulation. Arch. f. Exper. Path. u. Pharmakol., 1911, lxvi, 261–295.
17. Janossy, J. Ueber Wirkung intracisternoes verabreichter Medicamente: Coramin, Tropakokain. Deutsche Ztschr. f. Nervenh., 1930, cxiii, 278–287.
18. Jonnesco, T., and Jiano, A. L'anaesthèsie générale par injections intrarachidiennes. II Congréss de la Société Internationale de Chirugie, i, 282.
19. Koster, H., and Kasman, L. P. Spinal anæsthesia for the head, neck and thorax. Surg., Gynec. & Obst., 1929, xlix, 617–629.
20. Labat, G. Regional Anesthesia. p. 459. Philadelphia: W. B. Saunders Company, 1924.
21. La Mendola, S. Antagomismo tra . . . sulphonale, trionale, veronale, veronale sodico, luminale sodico e cocaina. Arch. di Pharmacol. sper. Sc. Aff., 1924, xxxvii, 256.
22. Martin, E. G., and Meyers, M. P. Detoxication of local anæsthetics. Tr. Am. Proctol. Soc., 1928, xxix, 82–88.
    Martin, E. G. Local anæsthetic agents, preventive and emergency treatment of toxicity with special reference to barbituric acid derivatives. J. Am. M. Ass., 1928, xci, 555.
23. Morrison, J. T. J. Spinal anæsthesia by tropacocaine ("the Ingleby lecture"). Brit. M. J., 1913, June, 1305.
24. Pauchet, V. Spinal anæsthesia. Am. J. Surg., 1920, xxxiv, 1.
25. Pitkin, G. P. Controllable spinal anæsthesia. Am. J. Surg., 1928, v, 553.
26. Rygh, E. A., and Bessesen, D. H. The causes of deaths from spinal anæsthesia. Minnesota Med., 1928, xi, 744–747.
27. Sise, L. F. Spinal anæsthesia fatalities and their prevention. New England J. Med., 1929, cc, 1072.
28. Soupalt, R. Dissociation des centres bulbaires par la novocaïne chez l'homme. Presse méd., 1923, xxxi, 379.
29. Tatum, A. L., Atkinson, A. J., and Collins, K. H. Acute cocaine poisoning: its prophylaxis and treatment in laboratory animals. J. Pharmacol. & Exper. Therap., 1925, xxvi, 325.
    Idem. Acute cocaine poisoning. Preliminary report of an experimental study. J. Am. M. Ass., 1918, lxxxiv, 1177.
    Tatum, A. L., and Collins, K. H. Acute cocaine poisoning and its treatment in the monkey (macasus rhesus). Arch. Int. Med., 1926, xxxviii, 405–409.
    Tatum, A. L. An experimental study of acute cocaine poisoning, its prophylaxis and treatment. Illinois M. J., 1926, xlix, 478–480.
30. Vehr, G. R. Heart beat and respiration in total novocaine analgesia. Northwest Med., 1931, xxx, 256, 322.

THE MECHANICS OF THE REVERSE FLOW OF BLOOD IN VARICOSE VEINS AS PROVED BY BLOOD PRESSURE READINGS

Its Clinical Application to the Injection Treatment

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MUCH has been written on the direction of the venous flow in varicose veins. Even so the subject is not settled in the minds of many men for sufficient proof has not been forthcoming to prove conclusively the true pathological situation existing.

THE NORMAL FLOW

The venous blood of the lower extremities is normally returned to the heart by means of the superficial and deep systems of veins. The superficial system is the one in which we are primarily interested at this time as in the great majority of cases the veins of the deep system with their valves remain normal and intact.

The superficial and deep systems of veins are connected by means of communicating veins. A large number of these are scattered throughout the lower leg and about the knee while in the lower thigh only a few are present and in the upper thigh often none exists. These communicating veins are also provided with valves which normally prevent the reverse flow of blood from the deep system outward.

From the time that varicose veins were recognized as pathological entities it has been the assumption that the upward flow of blood in them is markedly slowed and that varices remain as large, dilated tubes of blood.

A milestone was set in 1891 by Trendelenburg when he presented his classical thesis and paper in which he so clearly discussed the pathology of varicose veins that his conclusions are still accepted as accurate. The position in which the patient is placed while being studied still bears his name. Trendelenburg evolved certain tests that very clearly proved to him that the blood flowed downward from the saphenofemoral opening in certain cases and outward through the communicating veins in others.

Many authorities, Delbet and Mocquot, Hasebroeck and others, stated that in their opinion the deficiency of the valve at the saphenofemoral opening is the first step in the formation of varicose veins. This deficiency may be due to a degeneration of the valves with approaching age. In other cases there is no question but that it is due to a gradual widening of the vein lumen by a simple stretching of the vein walls which pulls the edges of the valves apart, thus allowing a true mechanical incompetency to develop though the valves themselves are not injured. In either of these events the blood would no longer be held in the vein above the valves but would flow in either direction according to the point of greatest hydraulic pressure.

These findings are clearly shown and proved by the simple application of the Trendelenburg test. When this test is made, the patient lies down with the foot and leg elevated high. The force of gravity would thus empty the varices and draw the blood into the general venous system. A tourniquet is tightly applied about the upper or middle thigh and the patient is allowed to stand. In any well developed case of varicose veins those veins above the tourniquet will rapidly fill but those varices below it will remain collapsed. If, when the tourniquet is suddenly removed, the lower varices fill rapidly the conclusion is drawn that they have filled with blood from above. This state of affairs is termed a Trendelenburg positive. When the test is made with the tourniquet tightly applied and the patient standing, if the varices fill quickly from below, then it is clear that the varices have filled from the deep system due to incompetent valves in the communicating veins. This condition is called a Trendelenburg negative. In some cases the varices will fill rapidly from below but when the tourniquet is removed they will fill still more tensely and then we have what is termed

a Trendelenburg double. By this we mean that the blood has come both outward through the communicating veins and downward through the great saphenous from the deficient valves above. Occasionally, the veins of the lower thigh and lower leg have become dilated and distended, yet all the valves are competent. In this case the varices would simply fill slowly with the returning blood from the distal parts of the extremity. Such a condition is called a Trendelenburg nil.

This pathological flow was very clearly demonstrated on several different occasions under the fluoroscope by means of lipiodol injections. It was also proved by Jentzer and other men abroad. A repetition of the experiment seemed to give more positive data than had been obtained before.

A patient with Trendelenburg positive varicose veins size 4 (2 centimeters in diameter), from groin to calf, was placed in the prone position on a tilting X-ray table. The leg was raised high to empty the varices. A tourniquet was then applied about the upper thigh just below a high varix. The table was then tilted bringing the patient erect. The large varix above the tourniquet rapidly filled while those below remained empty. Two cubic centimeters of lipiodol was injected into the distended loop above the tourniquet as shown in Figure 1. The tourniquet was then removed and the varicose system below filled with a gush carrying the lipiodol with it as shown in Figure 2. The lipiodol remained in position as in Figure 2 until the patient stepped back and forth, first on one foot and then on the other, thus contracting the calf muscles similar to that which would occur in walking. As this was done, the lipiodol rapidly disappeared into the deep system. This disappearance is explained as follows:

When the patient is standing still there is a constant upward surge of blood from the distal part of the extremity due to the *vis a tergo* being carried through the capillaries, the mild aspiratory effect of inspiration, and the increase of pressure on the deep veins by the contraction of the calf muscles which completely surrounds them.

In this discussion we will liken the muscles of the lower leg to those of the heart. The resultant action of the contraction of muscle groups in the leg is the same as in the heart, that is, to compress the contents and cause its expulsion through the patent openings into blood channels. In the heart these are the pulmonary artery and the aorta. In the calf they are the deep veins which pass upward and unite to form the deep femoral. During the period of relaxation, the deep veins of the lower leg are nearly empty and waiting to be filled the same as the chambers of the heart during the period of relaxation. For this reason it is very logical that we speak of the systolic and diastolic phase of muscular action of the lower leg inasmuch as it so accurately explains the normal functioning of the part and its action on the contents of the deep veins.

Let us accept this hypothesis as a basis for our reasoning. With the patient standing still, with all muscles tonic, the venous flow is upward similar to the overflow of a spring. Just as soon as this patient begins to walk the systolic contraction of the muscles of the lower leg empties the deep veins of the calf.

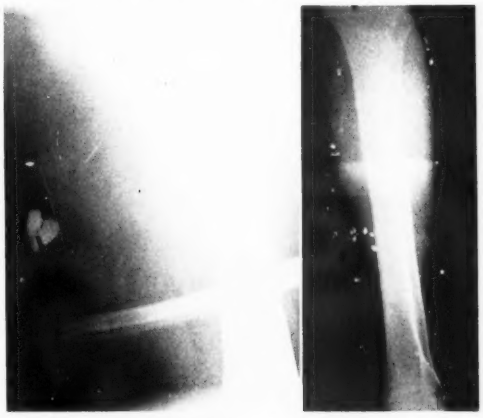

Fig. 1, left. Two cubic centers of lipiodol had been injected into a large distended varix, size 4, in the midthigh just above a tourniquet. The varices of the entire leg had been emptied previously by having the patient lie down with the foot in high elevation. The tourniquet was then applied in this position. Those varices above the tourniquet rapidly filled as soon as the patient stood up.

Fig. 2. The tourniquet has been removed allowing the lipiodol, as shown in Figure 1, to be carried downward with the blood in the distended varices above into the collapsed and comparatively empty varicosed segments of the lower leg. Note that some of the lipiodol has gone as low as the internal malleolus.

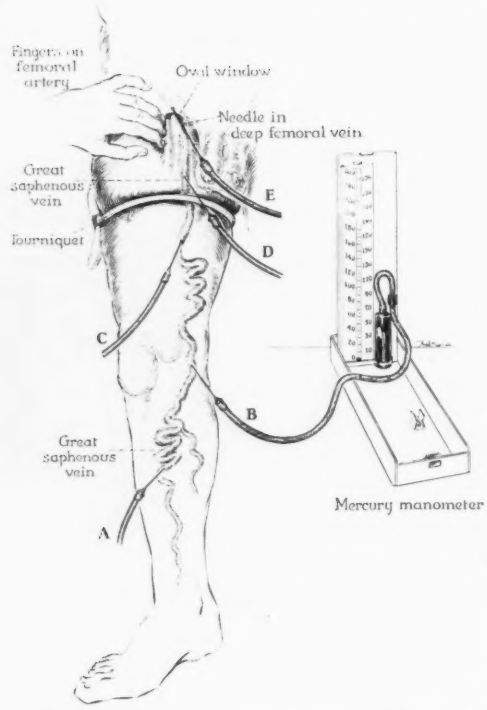

Fig. 3. Drawing showing the technical details of the experimental work on the blood pressure in varicose veins.

The competent valves of the deep veins maintain the column of blood above them, so that at the beginning of the diastolic period of the muscular action of the calf muscles the deep veins are empty as fluids tend to flow in the direction of the lesser pressure. At the beginning of the diastolic phase of the calf muscular action, the superficial varicose veins are filled and distended. The pressure in the varices of the calf at that time may be as high as 88 millimeters of mercury due to the weight of the column of blood in the distended vein up to the saphenofemoral opening and up to the heart. It is clear then to expect that the blood would flow through the communicating veins to fill the deep veins to a pressure equal to that in the superficial varicose veins. Temporarily the pressure in the superficial veins would drop slightly during this diastolic phase only to rise again, due to the reverse flow of

TABLE I.—THE BLOOD PRESSURE READINGS AS RECORDED IN AUTHOR'S CASES

	E	D	C	B	A
Prone	7.0	*	8.0	14.5	17.5
Standing—tourniquet on	30.0	*	13.2	22.0	40.7
Standing—tourniquet off	36.8	*	18.5	25.5	42.7
Five seconds	33.3	*	28.7	42.6	71.3
Ten seconds	35.6	*	34.0	56.6	72.3
Twenty seconds	35.6	*	40.0	64.6	73.6
Grunting	46.0	*	44.0	81.0	88.0
Stepping	42.0	*	36.0	36.0	61.3
Inspiration	42.0	*	32.0	?	?
Expiration	44.0	*	33.0	?	?

E, Deep femoral just above saphenofemoral junction; D, great saphenous; C, saphenous in upper thigh; B, internal knee; A, mid calf.
*Practically the same through as at E.

blood from above when the deep veins are filled, that is, during the systolic phase of the calf muscles.

This condition is most clearly seen and observed in a case with a marked *positive* Trendelenburg but with no *negative* Trendelenburg because the valves in the communicating veins would still be functioning and prevent an outward flow of blood from the deep system under the pressure of the systolic contraction of the calf muscles. If there were a marked *negative* Trendelenburg present then the valves in the communicating veins would be so deficient that they would not prevent this outward flow during the systolic period. On the contrary, the high venous pressure in the superficial varicose veins of the lower leg would be maintained due both to the reverse flow from the great saphenous above and the outward flow from the deep veins of the calf through the incompetent communicating veins. Due to this continuous stagnation and the high venous pressure the blood serum is forced outward through the walls of the veins and into the capillary spaces of the tissues and thus develops the state of œdema of the tissues. In this way we would have developed the state of supersaturation of the tissues of the lower leg. This occurs most about the lower third and ankle. Secondary to this poor and deficient circulation, the trophic state of the tissues becomes lowered and we have the *locus minoris resistentiæ* developed for a varicose ulcer to form following a slight injury or a hematogenous infection.

PROOF BY MEANS OF BLOOD PRESSURE READING

We undertook to prove the soundness of the theory described by means of simulta

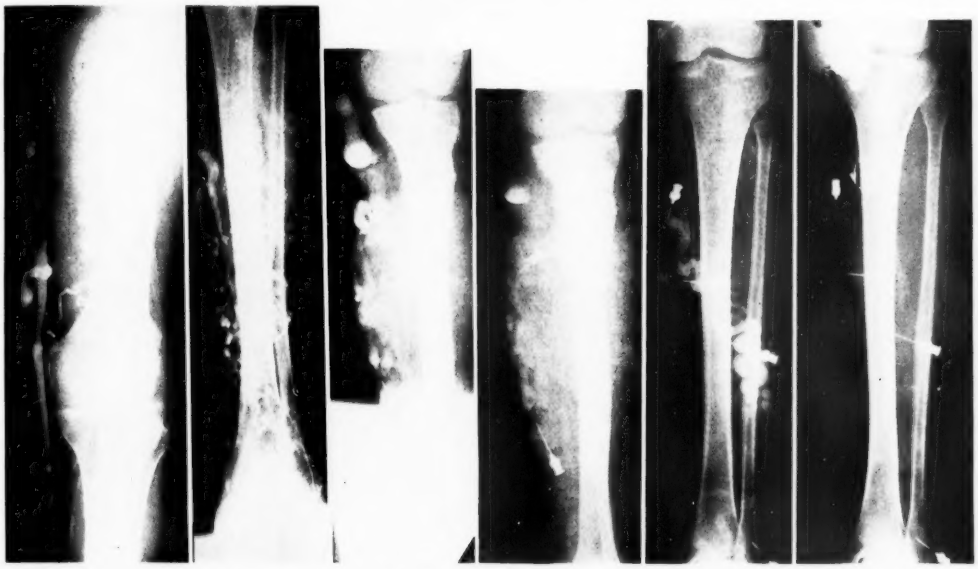

Fig. 4. Fig. 5. Fig. 6. Fig. 7. Fig. 8. Fig. 9.

Figs. 4 and 5. The skiodan was injected into the varicosed saphenous in lower thigh as described. It was carried downward into the varices of the lower leg. It then passed through communicating branches into the deep system where it was collected again into the popliteal vein and is shown passing upward to the femoral.

Fig. 6. This shows the skiodan mixed with equal parts of the usual salt and sugar mixture used in the injection treatment of varicose veins. This solution was injected into the varices of the calf and lower thigh exactly as is done when treating a case of varicose veins as described herein. The area of constriction is still shown below the knee where the tourniquet was applied in the effort to retain the solution locally. Another tourniquet was applied in the lower leg and a third in the lower thigh. The latter barely shows in the picture.

Fig. 7. The tourniquet has been removed and the patient has been stepping back and forth from one foot to the other, thus expelling the blood from the deep system. It will be noted that most of the skiodan has been drawn downward from the great saphenous and that in the calf it is much more diffuse and diluted. Further stepping washed the veins empty of the skiodan.

Fig. 8. Another case of varicose veins of the lower leg injected with the mixture of skiodan and sugar solution, equal parts. Tourniquets are shown below the knee and at the ankle. They clearly demonstrate how the injected solution can be retained locally.

Fig. 9. The solution is entirely disappearing following removal of the tourniquets and having the patient step back and forth from one foot to the other.

neous blood pressure readings at different points along the course of the varices of the great saphenous system (Fig. 3). We believed that the increase or decrease of blood pressure occasioned by the change of posture, prone or standing, would give us accurate data as to the direction of the venous flow inasmuch as the blood would flow strictly according to the rules of hydraulic pressure and fluids would tend to seek their level. This pressure would be due to the combined effect of gravity plus muscular action. The pressure in the superficial veins was also checked against that in the deep femoral just below Poupart's ligament (Table I).

The readings were taken at point A or mid calf; B, internal knee; C, saphenous in upper thigh; and E in the deep femoral just above saphenofemoral junction.

I will not bother you with tedious details of laboratory methods used but suffice it to say that from the readings shown in Table I, it is very clearly seen how the pressure in the great saphenous at *D* quite constantly approximated the pressure in the deep femoral which would mean that the valve controlling the reflux flow at the saphenofemoral opening was deficient and the flow of blood was in either direction, outward from the deep femoral or inward from the great saphenous ac-

cording to the direction of the greatest pressure either due to gravity or to muscular activity. The systolic effect of the calf muscles was beautifully demonstrated showing how the contractile force of these muscles during walking would force the blood upward through the great saphenous. This point is of great importance and interest in the study of the supportive "rubber sponge and Ace bandage" treatment of varicose ulcers. With the patient standing, the intra-abdominal pressure raised the pressure at all points quite considerably, meaning that the back pressure on all the vein valves was markedly increased. When the table was tilted and the patient brought to the prone position, the pressure rapidly dropped and when the leg was elevated the pressures were almost zero. This latter reading bears out clearly the effect of gravity in aiding the circulation and clearing away the œdema of the lower leg in cases of phlebitis and of ulcerations when the patient is put to bed with the leg in high elevation and hot packs.

To demonstrate visually the soundness of the conclusions drawn from the blood pressure tests, 20 cubic centimeters of a 40 per cent solution of skiodan was injected into the great saphenous vein at point *C*, with the patient standing erect and the varicosed system previously emptied as described (Figs. 4 and 5). Twenty cubic centimeters more of the solution was injected rapidly as the tourniquet was removed. The skiodan solution was carried downward and spread through the venous tree of the varicosed system exactly similar to its spread through the arterial system when injected there.

In the injection treatment of varicose veins it is essential, to obtain the best results, that we at all times keep in mind this reverse flow. It is our aim to bring the sclerosing solution into direct contact with the intima of the vein. This should at all times be controlled as much as possible as to the strength and concentration of the solution, its dilution by the blood, and its localization in the areas and segments of the veins, being treated. This can be accomplished by using the force of gravity to empty or fill the varices as desired combined with the use of tourniquets or occluders to retain and localize the injected solution. (Figs. 6, 7, 8, and 9.) The posture of the patient varies for the individual case although most patients are injected lying prone. At times the reverse flow is so marked that the injections are actually started with the leg in elevation while later it is lowered below the level of the table.

SUMMARY AND CONCLUSION

A. In any well developed case of varicose veins the blood is slowed or stagnant. When a patient is walking, the flow is actually reversed and is outward from the femoral and downward through the varicosed saphenous system. Proof of this statement is apparent with (1) the Trendelenburg and Perthe tests, (2) lipiodol injection of varicose veins, (3) skiodan injection of varicose veins, and (4) blood pressure experiments.

B. The theory of the reversed flow of blood in varicose veins and its mechanics should be applied in the treatment of every case. The actual technique employed in the individual case should vary with the case at hand.

C. The entire condition of one or both legs, should be treated completely at the first sitting to insure a better result. Theoretically there should be less danger of emboli formation with this technique. It is essential, also, that the saphenous vein be thrombosed up to the saphenofemoral ring to produce a lasting result.

D. As far as our search of the literature has revealed this is the first report of any work recording the blood pressure in the deep femoral in the human.

OBSTRUCTIVE PULMONARY EMPHYSEMA AND COLLATERAL RESPIRATION

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OBSTRUCTIVE emphysema of the lung was first described by Iglauer 20 years ago and has become well recognized since then by bronchoscopists and roentgenologists. Manges recorded 33 proved cases from his experience up to 1922. Jackson has written particularly extensively on the subject. This type of emphysema is clinically important because of its alarming effects and because its presence and distribution give reliable evidence of the existence and location of bronchial obstruction.

The purpose of this paper is to point out certain newly studied characteristics of the condition, particularly the tendency of the emphysema to result only from lobar bronchial obstruction. The cause for the failure of lobular bronchial obstruction to produce emphysema is assigned to collateral respiration, and that function of the lungs will be explained. The various other aspects of obstructive emphysema also will be discussed briefly.

ETIOLOGY

Iglauer and Jackson have observed bronchoscopically in man that a foreign body or neoplasm in a bronchus may produce valvular obstruction of a sort which permits air to pass only during inspiration. In some cases the object rolls or flaps with the air currents against an isthmus situated centrally to it in the bronchial lumen. In others the object is stationary and the respiratory movements of the bronchial wall produce the valvular action; for, with the dilatation of the bronchus that occurs at inspiration, the lumen opens slightly on one side of the object, and, with the contraction that takes place at expiration, the lumen closes tightly around it. Wessler and Jaches have described a case with obstruction by pressure from an enlarged peribronchial lymph node, where presumably the respiratory movements of the bronchus also affected the valvular action. In any case, the action brings about superabundant accumulation of air in the obstructed lobe or group of lobes and emphysema of those parts. The emphysema usually remains until the obstruction is relieved (for 3 months in one instance), and then it quickly disappears. However, emphysema may appear only during periods of exertive breathing; and it is always accentuated by this exertion. In most reports the bronchus in which the obstruction was found is not explicitly named, but the distribution of the emphysema is given and is said to correspond exactly to the territory supplied by the obstructed bronchus. One entire half of the lungs is reported to have been emphysematous in the majority of cases and single lobes in the rest, the right side being the more frequently involved. Careful search of the literature fails to disclose an instance with only a fraction of a lobe emphysematous.

SYMPTOMS

The development of emphysema is marked by a distressing sense of suffocation. Dyspnœa, cyanosis, and tachycardia appear and may become very pronounced, particularly when the patient exerts himself unduly to breathe, as in struggling and crying. Then death may result. The symptoms tend to subside during sleep or other periods of relaxation. Additional symptoms relate to pulmonary infection, tumor metastasis, etc., rather than to the emphysema, and are not pertinent to the present account.

DIAGNOSIS

Recognition of the nature of the condition is assisted by a history suggesting cause for bronchial obstruction, by the symptoms, and by certain physical and roentgenological signs. Physical signs include limitation of costal and diaphragmatic movements, hyperresonance to percussion, and reduction of breath sounds, on the side of the chest with emphysema;

also, exaggeration of respiratory movements and breath sounds on the other side. The heart is found displaced away from the emphysematous lung. Rarely a clicking sound produced by the valve is audible. Fluoroscopic inspection reveals increased size and radiolucence of the affected lobe or lobes, with displacement of the heart and depression of the hemidiaphragm. The ribs and diaphragm on that side move during respiration little or none at all, while those on the other side move exaggeratedly. The heart has a peculiar pendulous motion, swinging toward the affected side with inspiration and away from it with expiration. The difference in radiolucency and the visceral displacements are also to be seen in the roentgenogram; and they are especially pronounced if the film is exposed at expiration. The alterations in diaphragmatic, costal, and cardiac movements are represented in the film as well, if the double exposure method of Jung and Van Allen is used. Acute compensatory emphysema, as for instance that which occurs with massive pulmonary atelectasis, is differentiated from this form principally by the circumstances in the former that the diaphragm and ribs move more extensively on the side of the emphysema than on the other side and that the heart swings toward the emphysematous lung at expiration and away from it at inspiration. There is no other diagnostic roentgenological difference than this in many cases.

TREATMENT

Respiratory sedatives are given and the patient is kept as quiet as possible until bronchoscopic examination is made. The obstruction is relieved at the bronchoscopic examination by removal of the occluding body or by dilatation of the lumen. The therapy of the pulmonary infection and other complications of the bronchial obstruction is beyond the scope of this writing.

EXPERIMENTAL STUDIES

Obstructive pulmonary emphysema was produced in dogs by Hoover, in 1922. He introduced into the right primary bronchus a metal ring containing a valve. This permitted the passage of inspired air only, and after a few respirations the entire right lung became enlarged and emphysematous.

Lindskog and Van Allen recently studied the aerodynamics and other mechanical principles of bronchial obstruction of all types in dogs. Some of their data pertain to valvular obstruction of this type, and here the experiments were conducted as follows, briefly.

The animals were first anæsthetized and tracheotomized. A long slender cannula of special design was introduced through the trachea into the stem bronchus of the right lower lobe and was fixed there by dilating its end. The bronchial tree of the entire lobe was thus extended separately to the outside. Valvular obstruction was applied by connecting the cannula to a water-valve of the kind illustrated in Figure 1, at *W*. A manometer was also connected to the cannula. A second manometer was connected by another cannula to the pleural cavity. The breathing was rendered light and regular by carrying the animal in deep surgical anæsthesia. For about 10 minutes at the beginning it was noted that air entered quite abundantly through the valve at every inspiration and that the pressure within the obstructed bronchi rose progressively. Then the rate of admission of air declined rapidly until very little air entered, only one or two bubbles every three or four breaths, and this rate remained unchanged; also, the intrabronchial pressure ceased to climb and held to the elevated position it had reached. The intrapleural pressure thus far showed no appreciable alteration. After observing the situation for 1 hour and noting no change in these factors, the dog was caused to breathe deeply by lightening the anæsthesia to the excitement stage. This brought about a marked increase in the amount of air entering at the valve and rapid elevation of both intrabronchial and intrapleural pressures. After a few respirations the expiratory intrapleural pressure had reached nearly to atmospheric pressure. Then, although deep breathing was continued, the rate of entry of air decreased as before and the pressures remained at their new levels. These conditions showed no further tendency to change. After about 20 minutes, the anæsthesia was made deeper and the breathing lighter. Air stopped entering at once and the pressures began gradually to fall, until after a few minutes the intrabronchial pressure had descended to its first elevated level and the intrapleural pressure had reached normal. Air then began again to pass through the valve, one or two bubbles every third or fourth breath, and conditions became stationary once more. The dog was killed by injecting ether intravenously. With complete muscular relaxation of death, both pressures rose immediately to much higher points than those previously obtained, the intrapleural pressure being well above that of the atmosphere. Autopsy showed the right lower lobe greatly overinflated, the right half of the

diaphragm depressed, the heart displaced markedly to the left, and the other lobes correspondingly compressed.

It was concluded that emphysema is produced by lobar valvular obstruction of this type; that the degree of overinflation of the lung is determined directly by the depth of breathing and is stationary with a fixed respiratory depth; that after stationary conditions of inflation have been reached with a fixed respiratory depth, air enters the valve only in sufficient amounts to make up for absorption; that the pressure in the obstructed bronchi rises in proportion to the degree of overinflation; that the general intrathoracic (intrapleural) pressure is not altered unless extreme overinflation occurs; and that the relative stability of the general intrathoracic pressure is due to automatic accommodation by expansion of the thoracic parietes.

In other experiments of the kind, these workers introduced the cannula farther into the bronchus of the right lower lobe and fixed it at a point peripheral to the first branch of the lobe, so that lobular obstruction was obtained. Here the results were very different, for air entered abundantly through the valve at every inspiration for indefinitely long periods of time and the intrabronchial and intrapleural pressures suffered no mean elevation no matter how deeply the dogs were caused to breathe. Autopsy showed no overinflation of the lung. After many trials it was concluded that obstructive emphysema cannot be produced in a part of a lobe alone.

The fact that emphysema may be produced by lobar but not by lobular valvular obstruction has also been demonstrated roentgenographically, in new experiments.

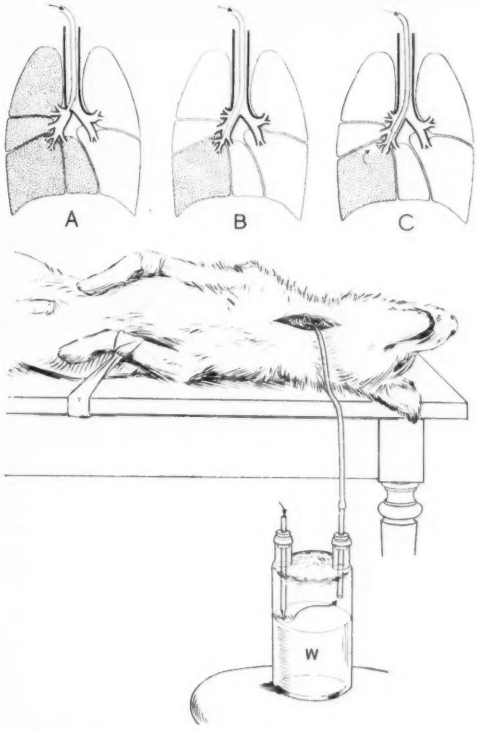

Fig. 1. Experimental method for producing valvular bronchial obstruction, as described last in the text. Below, Sketch of the anæsthetized and tracheotomized dog with the tube from the bronchial cannula emerging from the trachea and extending to the obstructing water valve, W. Arrows indicate the direction of flow of the respired air through the valve. Above, A, B, and C: diagrams of the lungs of the three dogs used, showing the points of fixation of the bronchial cannula and the areas of lung (dotted) so obstructed. Arrows above indicate the direction of flow through the tube and cannula. A, Lobar obstruction, including the entire right lung; B, lobar obstruction, including the entire right lower lobe; C, lobular obstruction, including about nine-tenths of the right lower lobe. Arrow below shows the collateral path of escape of air from the obstructed lobules.

Each of three dogs was anæsthetized and tracheotomized. A slender rubber tube, tipped with a short glass cannula, was introduced into the right primary bronchus. Positive pressure breathing was then administered and the chest was opened on the right by intercostal incision. In the case of one dog, the right primary bronchus was then isolated and encircled with a ligature at a point just central to the branch leading to the upper lobe. By tying the ligature the bronchus was fixed to the glass tube within it, and the entire right lung was thus separately cannulated (see Fig. 1, A). With the second dog, the stem bronchus of the right lower lobe was encircled with a ligature, the tube was maneuvered to that position, and the ligature was tied, so that the whole lobe was separately cannulated (see Fig. 1, B). With the third dog, the stem bronchus of the right lower lobe was dissected free of parenchyma far enough toward the periphery so that a ligature could be passed around it at a point peripheral to the first branch of the lobe. The tube was maneuvered to that place and the ligature was tied. This produced separate cannulation of more than nine-tenths of the lobe, leaving the remaining fraction free (see Fig. 1, C). After ligation, in each case, the lungs were fully expanded to exclude pneumothorax

A B C

Fig. 2. Roentgenograms of the chests of the three dogs (Fig. 1), showing the effects of valvular bronchial obstruction for 1 hour. A and B, Lobar obstruction, with emphysema of the entire right lung and of the right lower lobe respectively. Note the displacements of the heart and right hemidiaphragm. C, Lobular obstruction, without emphysema of the obstructed part (nine-tenths of the right lower lobe). Note the normal positions of the heart and diaphragm.

and the chest was closed. Artificial breathing was then stopped and a roentgenogram was taken. This showed the chest to be normal except for the presence of the tube and slight distortion of two ribs at the wound. The outer end of the tube was now connected with a water-valve (see Fig. 1). By lightening the anæsthesia the dogs were caused to breathe deeply. In the cases of the first two dogs (lobar obstruction), air entered the valve abundantly with every inspiration, for a few minutes. Then the rate of entry fell off and soon became constant at three or four bubbles of air per minute. X-ray examination at this time revealed the characteristic signs of obstructive pulmonary emphysema, involving the whole right lung in the first dog and the right lower lobe in the second. The roentgenograms are reproduced in Figure 2, at A and B. With the third dog (lobular obstruction) air entered the valve abundantly and without any reduction in rate. After 1 hour, X-ray examination showed no evidence of emphysema (see Fig. 2, C). The dogs were sacrificed and the roentgenographic findings were proved to be correct at autopsy.

The explanation for these differences in the effects of lobar and lobular forms of valvular bronchial obstruction evidently lies in the fact recently demonstrated by Van Allen, Lindskog, and Richter, that collateral connections exist between the airways of the lobules throughout the lobe but not between the airways of adjacent lobes. In their investigations the lobular units of the bronchial tree were found not to be independent of each other at the periphery as has been hitherto commonly supposed, but to be joined together by minute openings between the alveoli where the interlobular septa are incomplete. It was discovered that these connections are utilized to convey air during respiration to and from a division of a lobe when the bronchus of that part is totally obstructed. The function is termed collateral respiration. Ch'in and Van Allen and Van Allen and Jung demonstrated in both man and dog that one of the rôles of collateral respiration is to prevent atelectasis after bronchial obstruction. Atelectasis failed to develop with total obstruction of a lobular bronchus even after long periods of time, whereas total obstruction of a lobar bronchus caused atelectasis within 24 hours. Collateral respiration was found to fail and atelectasis to be produced with lobular obstruction only when the parenchyma of the free lobules in the same lobe was consolidated from pneumonia or other cause. The existence of collateral interlobular connections explains well the situation in regard to valvular bronchial obstruction that has been set forth

above; for in lobular obstruction the air which enters by the bronchial valve with each inspiration is free to leave the obstructed lobules collaterally with each expiration, without altering the degree of pulmonary inflation, while in lobar obstruction collateral escape is not provided for, the air accumulates, and overinflation results.

Referring again to the clinical aspects of obstructive pulmonary emphysema, it becomes clear why the condition has been seen only with lobar distribution. Furthermore, it is evident that the presence of normal inflation of the lung cannot be taken in any case as a guarantee of unobstructed bronchi. Also, the practice of keeping the patient from undue exertion in breathing until the obstruction is removed receives theoretical support.

SUMMARY

The etiology, symptomatology, diagnosis, and treatment of obstructive pulmonary emphysema are briefly discussed. It is noted among other points that in all cases which have been reported the distribution of the emphysema was lobar. Animal experiments are described which show that the condition can be reproduced in one or more whole lobes of the lung but not solely in lobular divisions. The latter fact is accounted for by reference to collateral respiration. Collateral respiration is explained and its general significance is discussed. Other experimental data are recounted to bring out additional characteristics of obstructive emphysema, particularly the fact that the overinflation of the lung is proportional to the depth of breathing and is not otherwise progressive. The relation of these facts to clinical diagnosis and treatment of the condition is indicated.

REFERENCES

1. Ch'in, K. and Van Allen, C. Permanent lobular bronchial obstruction in man without atelectasis. Ready for publication.
2. Hoover, C. Tracheal and bronchial stenosis as causes for emphysema. Arch. Int. Med., 1922, xxix, 143.
3. Iglauer, S. Three cases of foreign body in the bronchi. Lancet-Clinic, 1912, cvii, 603.
4. Jackson, C. A new diagnostic sign of foreign body in the trachea or bronchi, the "asthmatoid wheeze." Am. J. M. Sc., 1918, clvi, 625.
5. Idem. The symptomatology and diagnosis of foreign bodies in the air and food passages. Based upon a study of 789 cases. Am. J. M. Sc., 1921, clxi, 625.
6. Idem. Mechanism of physical signs in neoplastic and other diseases of the lung. J. Am. M. Ass., 1930, xcv, 639.
7. Jackson, C., Spencer, W., and Manges, W. The diagnosis and localization of non-opaque foreign bodies in the bronchi. Am. J. Roentgenol., 1920, vii, 277.
8. Jung, T., and Van Allen, C. A method of recording respiratory movements on a single roentgenogram. Nat. Med. J. China, 1931, xvii, 195.
9. Lindskog, G., and Van Allen, C. The aerodynamics of bronchial obstruction. Arch. Surg. (in press).
10. Manges, W. The roentgen-ray diagnosis of non-opaque foreign bodies in the air passages. Am. J. Roentgenol., 1922, ix, 288.
11. Van Allen, C., Lindskog, G., and Richter, H. Gaseous interchange between adjacent lung lobules. Yale J. Biol. & Med., 1930, ii, 397.
12. Idem. Collateral respiration. Transfer of air collaterally between pulmonary lobules. J. Clin. Invest., 1931, x, 559.
13. Van Allen, C., and Jung, T. Postoperative atelectasis and collateral respiration. J. Thoracic Surg., 1931, i, 1.
14. Wessler, H., and Jaches, L. Clinical Roentgenology of Diseases of the Chest. p. 50. Troy, New York: Southworth Company, 1923.

CARCINOMA OF THE GALL BLADDER AND BILE DUCTS[1]

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FROM 1907 to 1930, inclusive, 22,365 operations were performed at The Mayo Clinic on the gall bladder and biliary tract. In 15,422 of these cases stones were present either in the gall bladder or in the ducts. In the same period, 312 operations were performed for malignant conditions of the gall bladder or ducts; 100 of the carcinomata were in the ducts, and 212 were primary in the gall bladder. The incidence of malignancy in the gall bladder or ducts in the cases in which operation was performed for any condition of the gall bladder or biliary ducts was 1.4 per cent.

AGE AND SEX

In our series of 212 cases of primary carcinoma of the gall bladder, 157 (74 per cent) occurred among women. Zenker reported 72.9 per cent among women, Naunyn 83 per cent, and in Musser's series of 98 patients, 75 were women. Smithies reported a greater frequency in men, the proportion being 16 to 7.

Carcinoma of the bile ducts is more common among male patients, although the difference is not so marked. Fifty-eight of our 100 cases (58 per cent) occurred among men. In Rolleston's series of 90 patients with carcinoma of the bile ducts, 55 were males and 35 were females. A similar incidence was reported by Devic and Gallavardin, who found the condition in 30 males and 16 females.

The average age of our 312 patients was 57.1 years. There is no appreciable difference in the age at which carcinoma of the gall bladder and of the extrahepatic ducts is observed. Both occur most frequently between the ages of 50 and 70 years. Of the 312 patients, 0.9 per cent were aged less than 30 years, and the age incidence by decades from that period was as follows: 30 to 40 years, 4.7 per cent; 40 to 50 years, 14.9 per cent; 50 to 60 years, 40.3 per cent; 60 to 70 years, 33.1 per cent; and 70 to 80 years, 6.0 per cent. The youngest patient was 23 years of age, and the oldest, 78 years.

CLINICAL PICTURE

There is no distinct clinical picture of carcinoma of the gall bladder or of the biliary ducts, for the symptoms presented are dependent, to a great extent, on the situation of the lesion, its extent, and the changes associated with it. The most frequent complaint is of jaundice, which in contradistinction to jaundice caused by extrinsic mechanical obstruction of the bile ducts, is usually associated with pain. Vague gastric symptoms, characterized by belching, bloating, or milder forms of indigestion, are frequently complained of. Pain is chiefly in the right upper abdominal quadrant. This pain may be projected to the back, between the scapulæ, or to the right shoulder, and in some instances it extends across the abdomen to the left side. Frequently it originates in the epigastrium, and is similarly projected. It may be localized in the lower portion of the abdomen, or in the right side of the thorax, but relatively rarely. The pain has no definite relationship to meals, nor is there any definite relationship to the quality or quantity of food. Pain at night is rare, although if the condition is associated with cholelithiasis, there may be some nocturnal distress. Morphine is not infrequently required to relieve the pain, particularly that of typical biliary colic, which undoubtedly is a result of the presence of stones rather than of the malignant process *per se*. Changing the posture, and the application of external heat to the region, are of little benefit in any case.

Lancereaux has divided cases of malignancy of the gall bladder into a biliary form, characterized by belching, abdominal cramps, dyspepsia, jaundice, presence of an abdominal mass, and fever, and an hepatic form, of insidious onset, short duration, and accompanied by vague abdominal pains, weakness, diarrhea or constipation, rapid enlargement of the liver, and occasional slight jaundice. Rolleston divided such cases into those in which the sym-

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toms are associated with pre-existing cholelithiasis, those in which the symptoms are due to the local effects of the disease, and those in which they are due to invasion of adjacent parts by the growth, and to metastasis in the liver, peritoneum, and elsewhere.

According to our experience, if the lesion is in the common bile duct, the onset of symptoms is insidious, and the first symptom generally is jaundice, which is usually accompanied by pain. The clinical picture is then one of obstructive jaundice, and varies with the situation of the growth and other associated conditions, such as infection, stones, or pancreatitis. There may be a sudden onset of rather typical biliary colic, which would suggest an impacted stone, but which may occur even in the absence of choledocholithiasis, associated cholangitis, or pancreatitis. Infrequently there are chills and fever, and a history of recurrent attacks is not uncommon.

In the 312 cases, the duration of the symptoms concerning which the patient complained in most cases was less than 6 months, and in one-half of these the symptoms had persisted for 2 months or less. In the remainder of the cases there may have been considerable distress in recurrent attacks for a period of years, but these were undoubtedly due to associated conditions rather than to the lesion in question. The degree of jaundice was usually marked, the concentration of bilirubin often reaching as much as 35 to 40 milligrams in each 100 cubic centimeters of serum. A definite clinical diagnosis was exceedingly difficult.

W. J. Mayo has mentioned the following points in connection with clinical diagnosis of malignant neoplasms in the gall bladder: a hard tumor in the region of the gall bladder, absence of rigidity unless the peritoneum is involved, progressive loss of flesh and cachexia, a nodular tumor if the liver is involved, and jaundice if the ducts are involved. In our series of cases, however, a palpable tumor in the region of the gall bladder was observed relatively infrequently, and most often the clinical diagnosis was cholecystitis with cholelithiasis.

The importance of early diagnosis cannot be overestimated. Pallin recently analyzed 52 cases of carcinoma of the bile ducts. Anastomosis was done in 9 cases, and in 7 the ducts were drained. Twenty-five of the 31 patients who were operated on died almost immediately. In one-half of the cases, deaths were attributed to postoperative hæmorrhage, and 2 to anuria. Injury from the bile in the blood was responsible for 18 of the 25 fatalities. Pallin stated that danger from cholæmia is seldom great until the jaundice has persisted for from 3½ to 4 weeks, and concluded, therefore, that operation should be performed in not more than 3 weeks after severe, persisting jaundice has been suspected of arising from malignant disease.

Fig. 1. Carcinoma of the cystic duct with obstruction of the common duct. Cholelithiasis. One stone impacted in the cystic duct.

SURGICAL TREATMENT

A satisfactory method of dealing with cases in which carcinoma of the gall bladder or biliary ducts is suspected is to institute an immediate short period of observation in hospital. During this time, duodenal drainage is

Fig. 2. Closer view of Figure 1. Occlusion of the common duct by the growth in the cystic duct.

Fig. 3. Same as Figure 1. Calculi have been removed to demonstrate the increased thickness of the wall and the trabeculation of the gall bladder. The malignant process has not extended above the cystic duct.

attempted, and solution of glucose and sodium chloride solution, with the addition of calcium chloride, is administered intravenously. Transfusion of blood once or twice prior to any contemplated operative procedure is of distinct benefit. If no bile is obtained after repeated efforts at duodenal drainage, and if the concentration of serum bilirubin remains stationary or shows any tendency to increase, that complete biliary occlusion has occurred is a logical deduction, and immediate operation must be done to re-establish adequate flow of bile. On the other hand, if bile is obtained through the duodenal tube, and the concentration of serum bilirubin shows a tendency to decrease, operation may be postponed until the value for serum bilirubin has become low and constant, and the general condition of the patient is as satisfactory as possible.

The surgical treatment of patients with malignant disease of the gall bladder or biliary ducts is either palliative or radical. Of the 312 cases studied in this series, exploratory operation, with removal of a small bit of tissue for diagnosis in a few cases, was the only surgical procedure carried out in 172 (55.1 per cent). In 59 cases it was possible to remove the gall bladder. In 42 cases cholecystostomy only was done, and in 9 the gall bladder was removed and the common bile duct drained at the same time. Anastomosis was made in 27 cases; in the majority of them, cholecystgastrostomy was carried out; cholecystduodenostomy was performed in 9 cases, and cholecystjejunostomy in 1 case. The ampulla of Vater was resected in 3 other instances.

The type of surgical treatment indicated depends entirely on the situation and extent of the growth. Immediate establishment of biliary drainage is essential and should be carried out before radical resection is attempted. A good example of what one may accomplish is given in a case of carcinoma of the ampulla of Vater which was observed years ago. The first operation in this case consisted of choledochostomy with drainage of the common bile duct for a period of

Fig. 4. Carcinoma of the gall bladder, with metastasis to the liver. Occlusion of the cystic duct and the common duct. Cholelithiasis.

months. Transduodenal resection of the ampulla was then done. Unfortunately, there was recurrence at the site of the original lesion, and obstruction to the biliary flow necessitated cholecystgastrostomy. The recurrent lesion continued to enlarge and finally produced duodenal obstruction, so that a fourth operation was necessary, at which time posterior gastro-enterostomy was performed. The final flow of bile in this case, therefore, was through the gall bladder and into the stomach where it united with the food, and from the stomach through the gastroenteric stoma.

Early lesions in the gall bladder, of course, necessitate cholecystectomy. Rather early metastasis occurs if the lesion is confined to the gall bladder, for the lymphatic drainage from this viscus is profuse. Springer advised for middle-aged persons who have complete and persistent obstructive jaundice, that cholecystenterostomy be performed first. Radical resection is performed after 4 or 5 weeks if such a procedure is possible.

Carcinoma of the cystic, common, or hepatic duct may be treated by resection of the involved parts, or by the palliative procedure of establishing biliary continuity by means of one of the methods of biliary intestinal anastomosis. Cholecystostomy only may be done to relieve infection and to decrease constriction in the duct below by lessening the spasm.

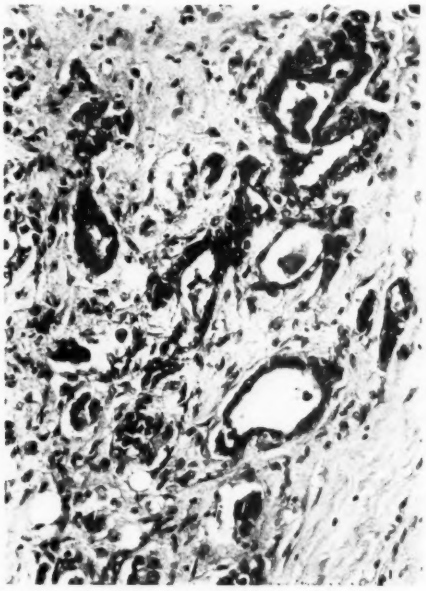

Fig. 5. Photomicrograph of a section of the wall of the gall bladder illustrated in Figure 4. Diffuse infiltration by islets of adenocarcinomatous tissue which has a markedly invasive character (×150).

CALCULI AS AN ETIOLOGICAL FACTOR

As little is known regarding the cause of malignancy of the gall bladder and the bile ducts as is known of carcinoma in general. The frequent association of gall stones with malignancy of these structures (Figs. 1, 2, and 3) has led many writers to believe that the chief causal factor is local irritation. In the 212 cases of carcinoma of the gall bladder that are reviewed in this report, stones were present in 137 (64.6 per cent). Interesting experimental work along this line has been done by several observers.

Kazama was the first to insert foreign bodies into the gall bladder in order to produce carcinoma. He experimented on the dog, rabbit, and guinea pig. Of the foreign bodies which he used, some had a purely mechanical action; among these were stones, sutures, and pieces of vesicular mucosa. He obtained from his research only the knowledge that by inserting small stones in the gall bladder of the guinea pig, in a relatively short time, papillomatous formations and adenocarcinomata

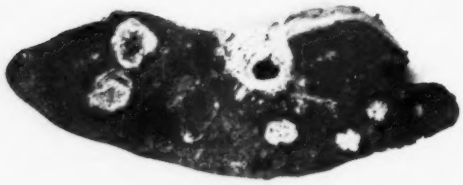

Fig. 6. Cross section of the liver through a region including carcinoma of the gall bladder and multiple metastatic growths in the liver.

with metastasis could be produced. In the 30 animals he produced 4 tumors, with metastasis in the thoracic wall, the intestine, and the liver. From this he concluded that the gall bladder of the guinea pig is an elective organ for the study of experimental carcinoma.

Leitch confirmed the results obtained by Kazama, and reported in detail his experiences. He inserted in guinea pigs, calculi, small sterilized stones, and pellets of tar. Of 35 animals operated on, 2 died quickly of hæmorrhage, 17 died after a period of 15 days to a year, and 15 lived even after 15 months. Results of necropsy of 2 guinea pigs are illustrative of the changes found. A year after insertion of the calculus the gall bladder of one animal was much thickened and adherent to the liver, and microscopically there was a layer rich in cells in which were buried tubular formations. At other points tubules were large, sometimes irregular, and covered with several layers of long cylindrical cells. In the second animal examined also a year after insertion of a small stone, there were perivascular adhesions to the liver, to the intestine, and to the omentum, and the gall bladder was transformed into a thick mass. Microscopically, the wall was covered with epithelium which had proliferated inward into dense connective tissue. At certain points the epithelium touched the vesicular cavity and was of the pavement type. The adherent omentum was infiltrated with carcinomatous cellules. Leitch estimated that he found carcinomatous lesions in 8 guinea pigs.

Barlow introduced a new factor in the pathogenesis of carcinomata of the gall bladder. He demonstrated that the calculi taken

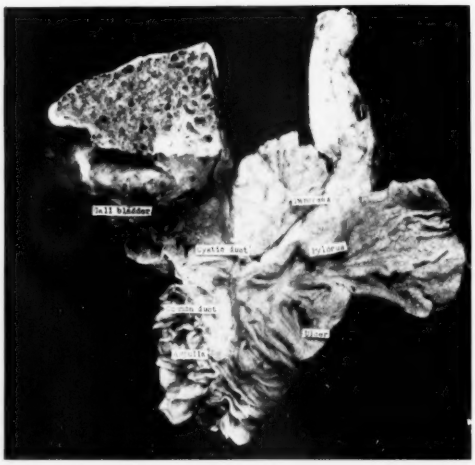

Fig. 7. Carcinoma of the gall bladder, with invasion of the common and hepatic ducts. Metastasis to the liver, cirrhosis of the liver, and healed duodenal ulcer.

from a carcinomatous gall bladder were radio-active, and he thought that this radio-activity was the cause of the carcinoma. Delbet and Godard have undertaken to verify the conceptions of Barlow. Calculi were taken from the carcinomatous vesicles of human patients and were submitted to examination by La Borde who was unable to discover any radio-activity. However, these two observers believed that other indeterminate factors may give to these calculi a peculiar cancerigenic power. In order to verify this they undertook two series of comparative experiments. In one series, calculi taken from gall bladders of man, some carcinomatous and others not, were inserted in the bladders of guinea pigs. In addition, part of the pigs of each series was submitted to injection of tar. Histological study of these gall bladders, after varying lengths of time, failed to produce the characteristic picture of carcinoma, and the conclusion that these two observers have made fail to confirm the findings of Kazama and Leitch.

PATHOLOGY

According to Ewing, carcinoma of the gall bladder appears in three main forms: (1) villous, papillomatous, or fungating, (2) gelatinous, and (3) diffuse, flat, and infiltrating.

Fig. 8. Carcinoma of the gall bladder, with metastasis to the liver. Obstruction of the common duct by carcinoma. Cholelithiasis.

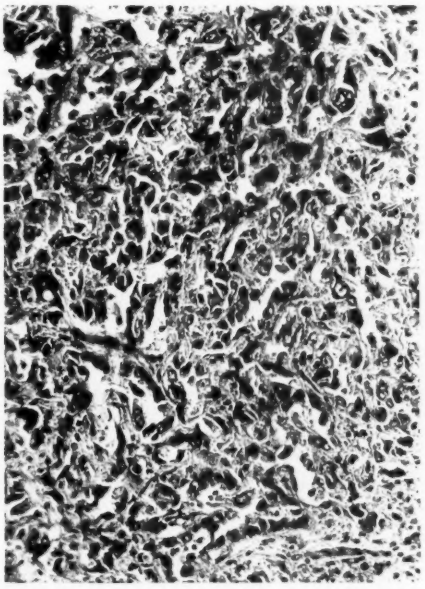

Fig. 9. Photomicrograph of a section of the bed of the gall bladder shown in Figure 8. Irregularly arranged and shaped cells; some are arranged in structures which roughly simulate acini. Numerous mitotic figures (×140).

The disease first appears as a papilloma, or as a flat induration, or an eroded ulcer, and the situation is usually at the fundus, the neck, or at the cystic duct. The papillary form grows out into the bladder as a coarse, villous, or solid fungating mass which eventually distends and completely obliterates the lumen of the gall bladder. According to this observer, early papillary tumors are rarely seen, but may appear as fragile, villous, or warty growths in a distended cavity. These may grow along the cystic duct into the hepatic ducts or into the common bile duct.

Gelatinous carcinomata infiltrate the wall, fill the cavity of the gall bladder, and early metastasis to the liver or direct extension occurs. Kaufmann stated that, disregarding the stomach, the gall bladder is the most common source of gelatinous carcinoma. Infiltrating tumors originate in the submucous tissues or as an ulceration in the mucosa. The wall of the gall bladder is infiltrated at an early period, and becomes greatly thickened and contracted. According to Rolleston, the majority of infiltrating tumors are of the scirrhous type, converting the gall bladder into a hard, contracted mass, without appreciable increase in bulk; extension to adjacent viscera is not uncommon. Metastasis is usually to the liver (Figs. 4, 5, 6, 7, 8, and 9), abdominal nodes, and peritoneum. Beadles and West have described extensive enlargement of mediastinal and supraclavicular nodes. Warthin stated that metastasis to the suprarenal glands may give rise to cutaneous pigmentation, suggesting that of Addison's disease.

Bland-Sutton stated that carcinoma may arise in the epithelium in any of the bile ducts in the liver, but that it more commonly arises in the excretory apparatus than in the intra-

hepatic system. When the disease arises in the small, intrahepatic ducts, it is, for practical purposes, primary carcinoma of the liver. According to this observer, carcinoma of the excretory apparatus of the liver is frequently seen in the hepatic duct, at the juncture of the hepatic and common bile ducts, in the common duct, and in the ampulla, but it arises with greater frequency in the gall bladder.

In 1921, Magoun and Renshaw reviewed the cases of malignant neoplasia in the gall bladder that had been observed in The Mayo Clinic since 1907. Within this period primary malignant growths, confirmed by pathological examination, occurred in 84 cases. Of these, 82 were carcinomata, 1 was an epithelioma, and 1 a lymphosarcoma. The following year Renshaw reviewed the cases of carcinoma of the extrahepatic bile ducts; during the same period as that of the preceding study, in 20 cases the diagnosis of primary carcinoma of the bile ducts was confirmed by pathological examination at The Mayo Clinic. Eight of the carcinomata were of the adenomatous type, 2 of the papillary type, and all had columnar, cuboidal, or spheroidal cells. Grossly, they varied from the annular constricting type to the flat, diffuse, and occasionally villous type.

In the series we are studying, malignancy of the gall bladder was definitely proved in 165 cases. Carcinomata occurred in 140 cases, 2 of which were definitely described as being of the colloid type. A combination of squamous cell epithelioma and adenocarcinoma occurred in 15 cases, a papillary form of adenocarcinoma in 5 cases, and squamous cell epithelioma in 4 cases. As in the cases previously reported by Magoun and Renshaw, the fundus was no more frequently involved than was the pelvis. In one additional case, lymphosarcoma involved both the fundus and the pelvis.

Again in our series, in 52 cases the diagnoses of primary carcinoma of the bile ducts were confirmed by pathological examination. In 28 cases the lesion occurred in the common duct; the diagnosis in all of these cases was carcinoma, and in 3 of them the growth was of the papillary type. Nine cases of carcinoma of the ampulla were observed, 1 of which was colloid in nature and another papillary. Carcinoma of the hepatic ducts occurred in 8 cases, 1 of which was papillary, and in 7 cases the growths arose from the cystic duct, all of which were carcinomata.

In 1927, Webber made an effort to determine a relationship between the length of life of patients after operation, and the grade of malignancy of primary carcinomata of the gall bladders that were removed. Thirty cases of primary carcinoma of the gall bladder were studied, but 4 of the patients died in hospital, leaving 26 for further investigation. In 12 of these, in which the growths were graded 2 or less, the patients lived an average of 2 years and 10 months after operation. Fourteen patients with carcinomata graded 3 or more lived an average of only 4.8 months. Of 12 tumors graded 2 or less, 4 were found at operation associated with growths or microscopic evidence of extension or metastasis. Of 14 tumors graded 3 or more, 13 were found at operation to be associated with similar evidence of extension or metastasis. Of all cases of carcinoma of the gall bladder and biliary ducts observed in The Mayo Clinic, 65 per cent of those in which the specimen has been subjected to grading have been graded 3 or more.

SUMMARY

A pathological and clinical study of 212 cases of primary malignancy of the gall bladder, and 100 cases of malignancy of the extrahepatic biliary ducts, has been presented. This represents 1.4 per cent of all cases in which operation for lesions in the gall bladder or biliary tract were performed during the same period as that represented by the cases studied. Of cases of carcinoma of the gall bladder, 74 per cent occurred in women. Of cases of carcinoma of the bile ducts, 51 per cent occurred in men. The average age of our entire 312 patients was 57.1 years. Seventy-three per cent of them were between the ages of 50 and 70 years. The youngest patient observed was 23 years of age and the oldest 7[illegible].

The clinical picture of carcinoma of the gall bladder or biliary ducts is not distinct and depends entirely on the situation of the lesion and on associated conditions such as infection, the presence of stones, and pancreatitis. The

majority of patients present symptoms of less than 6 months' duration. However, in many instances symptoms persist for many years, and, during the whole period, disease of the biliary tract should be suspected. Of the symptoms, the most frequent is jaundice associated with pain in the right upper abdominal quadrant.

The surgical treatment of carcinoma of the gall bladder or biliary ducts may be palliative or radical. Exploratory operation only was possible in 55.1 per cent of the 312 cases. Cholecystectomy was performed in 59, cholecystostomy in 42, cholecystectomy and choledochostomy in 9, and an anastomotic operation in 27.

Stones were present in 64.6 per cent of the cases in this series in which the gall bladders were the site of malignant growths. Carcinoma occurred in 140 cases, squamous cell epithelioma and adenocarcinoma in 15 cases, a papillary form of adenocarcinoma in 5 cases, squamous cell epithelioma only in 4 cases, and lymphosarcoma in 1 case of 165 lesions of the gall bladder which were proved to be malignant. Of 52 lesions of the bile ducts, all of which were proved to be malignant, all were found, on histological examination, to be carcinomata, and the common duct was the most frequent site. In our series, 65 per cent of all growths which were graded proved to be of grade 3 or more. Of patients with tumors of the gall bladder graded 3 or more, in Webber's series, the average length of life was only 4.8 months. Carcinoma of the ducts or ampulla usually imposes a still more serious prognosis.

Whether stones may be an etiological factor in the production of malignancy of the biliary tract is not known. The high incidence of stones in association with this condition, however, cannot be discounted and presents an important factor in deciding for or against their removal when first observed.

The importance of early diagnosis of malignant lesions of the gall bladder or biliary ducts cannot be overemphasized. Since it is impossible to recognize a distinct clinical syndrome which may accompany malignant invasions of the biliary tract, the condition should be kept in mind, in order that treatment may be instituted while the disease is temporarily controllable, if not curable.

BIBLIOGRAPHY

1. Barlow. Quoted by Delbet and Godard.
2. Beadles. Quoted by Ewing.
3. Bland-Sutton, John. Tumors, Innocent and Malignant. London: Cassell and Co., 1922.
4. Delbet, Pierre, and Godard, Henri. Inclusion de calculs biliares humains dans la vésicule chez le cobaye. Bull. de l'Ass. franç. p. l'étude du cancer, 1928, xvii, 347–361.
5. Devic and Gallavardin. Quoted by Rolleston and McNee.
6. Ewing, James. Neoplastic Diseases. 2d ed., p. 694. Philadelphia: W. B. Saunders Co., 1922.
7. Kazama, Yoshiaki. The studies on the artificial production of tumors in viscera. Japan Med. World, 1922, ii, 309–312.
8. La Borde. Quoted by Delbet and Godard.
9. Lancereaux. Quoted by Ames, Delano. Primary carcinoma of the gall-bladder. Bull. Johns Hopkins Hosp., 1894, v, 74–80.
10. Leitch, Archibald. Gall-stones and cancer of the gall bladder. Brit. M. J., 1924, ii, 451–454.
11. Magoun, J. A. H., Jr., and Renshaw, Kinsley. Malignant neoplasia in the gallbladder. Ann. Surg., 1921, lxxiv, 700–720.
12. Mayo, W. J. Malignant disease involving the gall-bladder. In: A collection of papers published previous to 1909. Vol. i, 384–391. Philadelphia: W. B. Saunders Co., 1912.
13. Musser, J. H. Primary cancer of the gall-bladder and bile ducts. II. Primary cancer of the bile ducts. Boston M. & S. J., 1889, cxxi, 581–583.
14. Naunyn. Quoted by Sherrill, J. G. Cancer of the gall-bladder and ducts. Ann. Surg., 1906, xliv, 866–891.
15. Pallin, Gustav. Das Carcinom des Ductus Hepatico-Choledochus und seine chirurgische Behandlung. Beitr. z. klin. Chir., 1921, cxxi, 84–137.
16. Renshaw, Kinsley. Malignant neoplasms of the extrahepatic biliary ducts. Ann. Surg., 1922, lxxvi, 205–221.
17. Rolleston, Humphry, and McNee, J. W. Diseases of the Liver, Gallbladder and Bile Ducts. 3d ed., pp. 738–758. London: Macmillan and Co., 1929.
18. Smithies, Frank. Primary carcinoma of the gall-bladder: an analysis of twenty-three proven instances of the disease. Am. J. M. Sc., 1919, clvii, 67–74.
19. Springer, Ernest. New growths involving the terminal bile and pancreatic ducts. Boston M. & S. J., 1925, cxcii, 997–1000.
20. Warthin. Quoted by Ewing.
21. Webber, I. M. Grades of malignancy in primary carcinoma of the gall bladder. Surg., Gynec. & Obst., 1927, xliv, 756–760.
22. West. Quoted by Ewing.
23. Zenker, Heinrich. Der primaere Krebs der Gallenblase und seine Beziehung zu Gallensteinen und Gallenblasennarben. Deutsche Arch. f. klin. Med., 1888, xliv, 159–184.

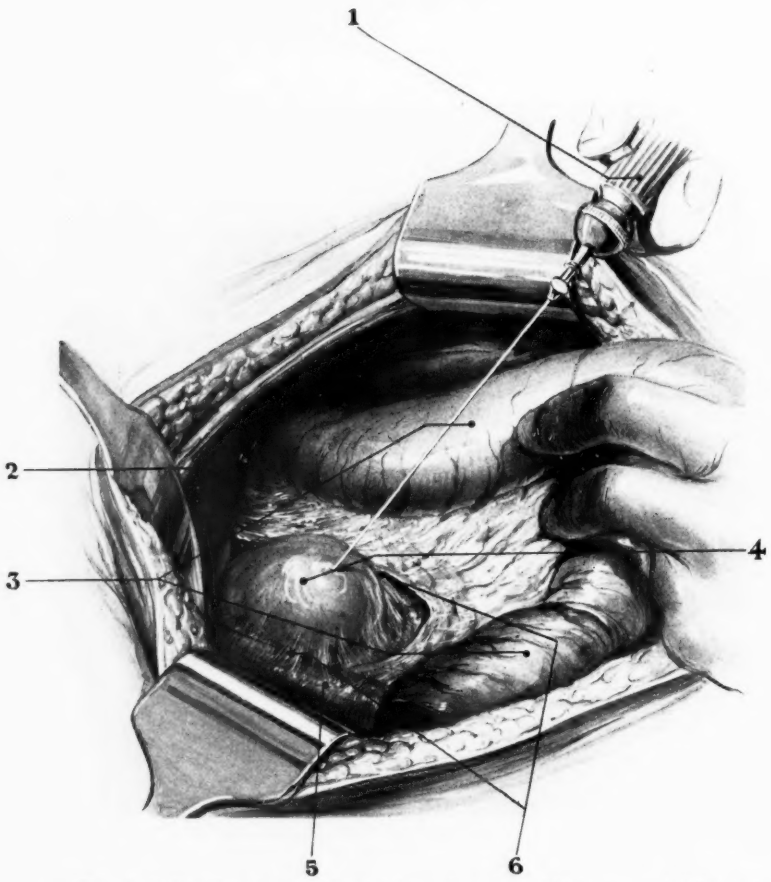

Fig. 18. Infiltration of retroperitoneal tissues by means of high pressure local anæsthesia method. Formation of an œdematous bleb under direct vision through a rent in the lesser omentum. 1, apparatus used for high pressure local anæsthesia; 2, liver; 3, stomach; 4, œdematous bleb; 5, vena cava inferior; 6, margins of wound in lesser omentum.

CLINICAL SURGERY

FROM THE SURGICAL CLINIC, UNIVERSITY OF TUEBINGEN

SPINAL ZONE ANÆSTHESIA

PLACED AT WILL AND DOSAGE INDIVIDUALLY GRADED[1]

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WHILE spinal anæsthesia is the method of choice in operations upon the lower extremities, it is within the abdominal cavity that it becomes the ideal procedure, unapproached by any other method because it affords unparalleled relaxation of the abdominal muscles, a quiet state of the patient, and an almost complete absence of postoperative complications.

Unfortunately, this inherently splendid method has been thus far regarded as a hit or miss procedure. It has not been possible to control the extent and the depth of anæsthesia. While the anæsthesia begins in the caudal spinal roots, it can be made through the increase of the dose or through the increase of the volume of the solvent or through changing the point of the spinal puncture to spread cranialward. One cannot, however, control either the level to be reached within the dural sac or hold it at the level attained. In one instance the anæsthetic solution will not spread sufficiently high cephalad and thus an incomplete anæsthesia may result, while in another it may spread too high and cause respiratory disturbances, a fall in the blood pressure, and collapse.

Still another shortcoming may be mentioned—the relatively great differences in the sensitiveness of individuals to a calculated dose, a fact which must not be disregarded. Each patient is given a predetermined average dose which may prove unnecessarily large in one case producing toxic symptoms or death, or it may prove too small in another case resulting in unsatisfactory anæsthesia. The dose will be exactly right for only a small proportion of cases.

The recently developed method of Pitkin has not obviated these disadvantages. The statement that spinocaine only because of its viscosity and lighter specific gravity will spread cranialward or caudalward when introduced into the dural sac, depending solely upon the position of the patient, is incorrect as anyone can at any time demonstrate upon a system of glass tubes. Spinocaine, when injected into the dural sac, can be seen to spread more often evenly to both sides without separating out because its specific gravity is different from that of the spinal fluid. I have not been able markedly to influence the spread of anæsthesia in patients either through the elevation of the pelvis or of the upper trunk. Were the proposition of Pitkin correct, it would certainly appear too dangerous—if only in the view of the variability in spinal curvatures—to depend, for the spread of so potent a solution, upon placing the diseased part or the entire body for a few minutes in an exactly prescribed degree of inclination. Pitkin's method does not discuss the question of individual dose.

We are using a new method of inducing spinal anæsthesia by means of which the anæsthetic solution can be placed at will in a definite segment of the spinal cord and maintained there; and in which the dosage is regulated to the individual patient much as it is in ether inhalation narcosis. These two conditions are arrived at in the following way:

1. With the patient placed so that the head is low and the buttocks are elevated, a spinal puncture is done and a certain amount of cerebrospinal fluid is removed and replaced by an equal volume of air. The air bubble collects in the highest portion of the dural sac within the sacral bone. An anæsthetic solution, which is lighter than the cerebrospinal fluid and being oil-like is not miscible with it, is injected into the dural sac. The fluid will naturally collect and take its place between the cerebrospinal fluid and the air bubble. Because it is lighter than the cerebrospinal fluid it cannot spread cephalad and it cannot pass toward the sacrum because of the lighter air.

In view of the fact, however, that the separation into three layers—the cerebrospinal fluid, the anæsthetic charge, and the air—will take place

[1] Translated by Dr. George Halperin.

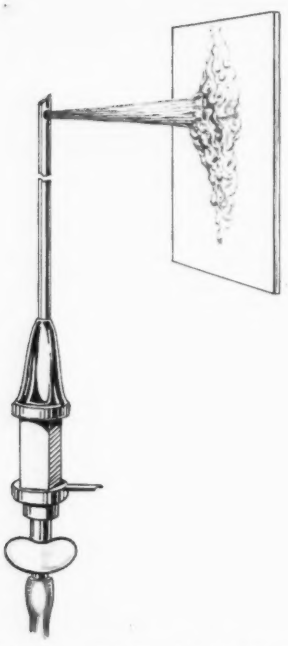


Fig. 1. Spinal puncture cannula with a lateral opening. The forcibly injected fluid is seen to diffuse transversely to the long axis of the cannula.

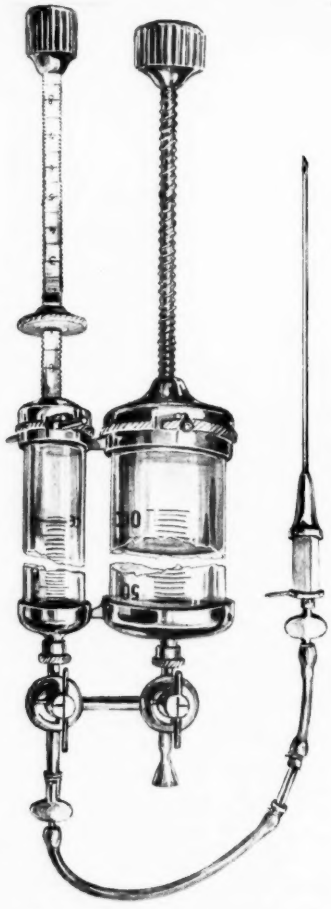


Fig. 2. The double syringe for spinal anæsthesia. The large syringe is for the aspiration of the cerebrospinal fluid and for the injection of air into the dural sac. The small syringe serves the purpose of injecting the anæsthetic charge. The stopcocks of the larger and smaller syringes control the outlets. The rubber tube and the cannula are joined by means of airtight bayonet pieces. The rubber tube is provided with a capillary glass tube so that the passage of air or fluid may be observed.

rather slowly and incompletely if dependent alone on the slight differences in the specific gravity, it is desirable to carry out still another procedure. This consists in injecting the anæsthetic solution through a special cannula the end of which is beveled off at 45 degrees and which is armed with a lateral opening just above the point (Fig. 1). The solution can be projected a considerable distance cephalad or caudad by turning the lateral opening in one or the other direction. Only the combination of this mechanical force plus gravity will insure a dependable separating out of the cerebrospinal fluid, the anæsthetic charge, and the air.

Only those spinal roots which pass through the layer of the anæsthetic solution will become anæsthetized. The roots corresponding to the layer of air or of cerebrospinal fluid will not be thus affected. In this manner we accomplish (1) *zone anæsthesia* (Fig. 10).

2. If to begin with insufficient air is injected or if some of it is aspirated, the anæsthetic solution will advance in the direction of caudal roots and the zone of anæsthesia will involve the lower extremities (Fig. 11). If considerable air is injected at the first or second attempt, the anæsthetic charge will be driven cephalad advancing the zone of anæsthesia in the same direction (Figs. 9 and 10) while the legs whose corresponding roots are protected by the area of air will retain their sensibility. In this manner we accomplish (2) a *spinal zone anæsthesia* limited to the upper trunk.

3. Rather than introduce the calculated dose at once, one may begin with a smaller dose, testing the skin sensibility after injection, and adding

more anæsthetic if necessary, thus securing the desired depth of anæsthesia in stages and giving each patient the minimum amount of the drug, neither more nor less! We thus secure (3) controllable *spinal zone anæsthesia with individually determined dose!*

APPARATUS[1]

To achieve these ends one requires special apparatus. The principal instrument is a double syringe (Fig. 2) consisting of a larger barrel of the capacity of 50 cubic centimeters for air and of a smaller barrel of 10 cubic centimeters' capacity for the anæsthetic solution. The outlet of both barrels is united by a crosspiece. Each is provided with a stopcock at the joint with the outlet of each barrel reaching beyond the stopcock. The extremity of the larger barrel expands in the form of a triangle so that nothing can be attached to it. To the end of the smaller syringe a tube armed with a bayonet metal piece may be attached. To the other end of the tube is attached the spinal puncture needle. The stopcock of the larger barrel controls (1) its own outlet and (2) that of the crosspiece. The smaller syringe stopcock controls (1) its own outlet and (2) the connection between its outlet and the crosspiece. By turning the cock one can therefore connect the larger barrel with the outside or with the puncture cannula or in the second place one can disconnect the smaller barrel from the cannula.

The piston of the larger barrel runs in a spiral groove and can be slowly moved by turning the screw. The piston of the smaller barrel is freely movable but can be set by means of a screw attachment so as to make it impossible to expel more than the desired amount of its contents.

A rubber tube 20 centimeters long is attached to the smaller syringe by means of a bayonet stopcock. Close to the other end the tube is interrupted by a short glass tube and at its end is armed with a bayonet-like metal piece which is prolonged as the special puncture cannula. The tube is strengthened at all joints by metallic bands guarding it against slipping even under considerable pressure. The special puncture cannula is 10 centimeters long, has an outer diameter of 1.1 millimeters, and tapers off at the bevel of 45 degrees. It is armed with a lateral opening just above its point.

The pistons, the stopcocks, and all of the joints are watertight so that no air or fluid can escape. To make them even more secure all the joints after boiling are greased with sterile vaseline before they are used. Because of the great importance of its being leak proof the entire system should be thoroughly tested from time to time, under water. Failure to do so is likely to lead to poor results.

[1] Made by the Jetter-Scheerer Company.

Fig. 3. Instruments and apparatus for the induction of spinal anæsthesia.

TECHNIQUE OF INDUCING SPINAL ANÆSTHESIA

The method is carried out in the following manner: The patient receives 0.05 grain of ephetonin (Merk Dormstadt) instead of morphine just before the induction of spinal anæsthesia. In the case of an abdominal operation a vasano suppository (Schering-Kahlbaum, Berlin) is given one hour before. This can be augmented later by a subcutaneous injection of vasano should nausea ensue. A table is set up for the anæsthetist as follows (Fig 3): (1) a small dish with alcohol sponges for the preparation of the patient's back for the spinal puncture; (2) a 10 cubic centimeter record syringe with a slender needle for the induction of a dermal wheal and the thicker one for the infiltration of the interspinous area; (3) 10 cubic centimeters of one-half per cent novocain-suprarenin solution for the above infiltration; (4) a three edged steel trocar for the puncture of the skin and of the interspinous ligament; (5) two special puncture needles, 1.1 millimeters in diameter, armed with a lateral opening; (6) a 50 cubic centimeter graduated glass cylinder for the reception of the aspirated cerebrospinal fluid; (7) two three minute hour glasses with an attached steril-

Fig. 4. Position of the patient while the anæsthesia is being inducted. The body lies with the pelvis elevated at an angle of 25 degrees to the horizontal plane. Slipping from the table is prevented by a strap. The double syringe is connected with the spinal needle.

izable handle; (8) a freshly sterilized special double syringe with an attached rubber tube the joints of which are greased with pure sterilized vaseline. The two barrels are filled in the following way: My percain charge is aspirated from ampuls into the smaller syringe. The larger cylinder with the stopcock open is placed at 30, so that its effective part now contains 20 cubic centimeters of air. The stopcocks are turned so that the larger barrel is connected with the puncture cannula.

The anæsthesia is inducted on the operating table; draping the patient on a separate table would prove inconvenient.

The patient is placed in a lateral posture (Fig. 4) with the long axis of the body at an inclination of 25 degrees to the horizontal plane, head low, buttocks elevated. The operating table is provided with an indicator or with a protractor attached to its axis, by means of which the degree of inclination can be accurately told. The slipping of the patient from the table is prevented by the use

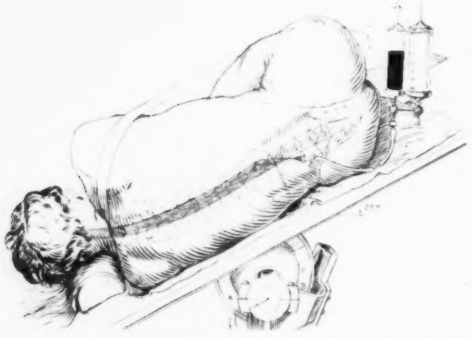


Fig. 5. The caudal portion of the dural sac is filled with air. The liquor level corresponds with the point of the entrance of the needle.

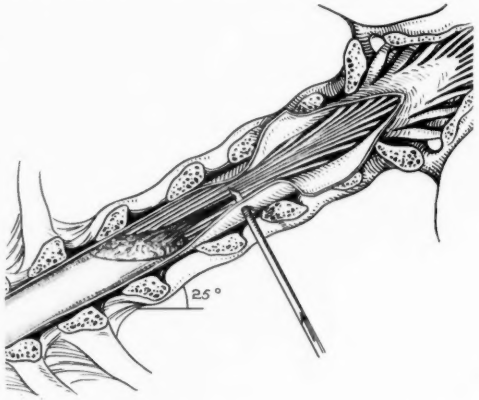


Fig. 6. "Head shot." The anæsthetic charge is ejected cranialward through the lateral opening in the cannula turned in that direction. Because of its lighter specific gravity and because it is not miscible with the cerebrospinal fluid, the "charge" collects at the periphery of the cerebrospinal fluid and below the air bubble.

of a special strap. A special assistant supports the patient and keeps his back flexed. The inclination of at least 25 degrees must remain unchanged during the induction of anæsthesia, during the operation, and after the operation until the anæsthesia has worn off! A special assistant who watches the pulse, respiration, and blood pressure and who attends to the wishes of the patient, is likewise charged with the responsibility of keeping the patient in the oblique position even during a change of posture, as for example when draping the patient for the operation either on his back or the abdomen, or in transporting him to his bed. For even a momentary raising of the head can send the air and the anæsthetic charge with it cranialward. When the area of operation is confined to one side of the body, the patient is placed during the induction of anæsthesia in an inclined position with that side up, for frequently anæsthesia spreads faster to the upper half.

The technique of induction of the anæsthesia varies, depending on whether a high anæsthesia involving the abdomen is desired, or low from the symphysis down. The anæsthetic solution should be directed cranialward from the point of entry of the needle in the lumbar region. For low anæsthesia the same should be directed caudalward. We differentiate in high anæsthesia still further between anæsthesia of the upper abdomen (operations upon stomach, bile tracts, transverse colon, spleen) and that for the lower abdomen. In low anæsthesia we differentiate between that for extremities and that for the perineal region (operations upon the prostate, anus, and the perineum).

a. *Technique of high spinal anæsthesia.* Lumbar puncture here is made as high cranialward as possible, in order to expedite the placing of the solution in the dorsal segment of the cord. Because of the possibility of injury to the cord, however, it is never introduced higher than the interspace between the first and second lumbar vertebræ.

As soon as the flow of the cerebrospinal fluid is established the double syringe with the larger barrel set at 20 cubic centimeters of air and the smaller filled with 10 cubic centimeters of percaine solution, is attached to the puncture needle by means of its rubber tube (Fig. 4). From now on the syringe is never detached from the needle until the completion of the induction of the anæsthesia. The stopcock between the large barrel and the cannula is opened and the cerebrospinal fluid is aspirated into it by a backward turn of the piston. For operations in the upper abdomen (stomach, bile tracts, spleen) 25 cubic centimeters are withdrawn, while for the anæsthesia of the lower abdomen about 20 cubic centimeters are removed. A lesser amount will also suffice, a resulting excess of 10 or even 20 cubic centimeters of air will do no harm.

The stopcock of the large syringe is closed and the aspirating fluid is emptied into a measuring glass. Its piston is set at the zero mark so that the syringe will contain 50 cubic centimeters of air. The stopcock is turned so as to connect the large barrel with the cannula. From now on the stopcock is no longer disturbed. The lateral opening of the puncture needle corresponding to an indicator at its head is directed toward the buttocks.

Air is injected into the dural sac by turning the piston of the large barrel. Upon injection of 10 to 12 cubic centimeters of air more cerebrospinal fluid is aspirated by turning the piston in the

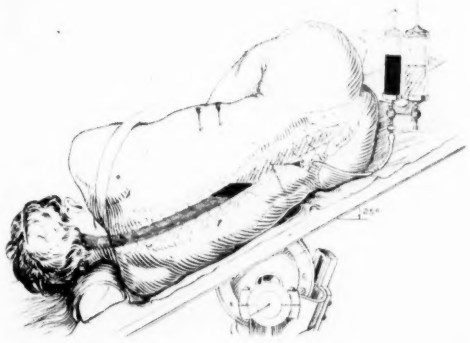


Fig. 7. Additional injection of air has forced the charge into the region of the lower thoracic vertebræ. The cranial limit of the anæsthetic zone as tested by pinpricks has been demonstrated to be at the level of the navel.

Fig. 8. Too much air was added. The charge has almost reached the region of cervical vertebræ. The zone of anæsthesia reached to the neck.

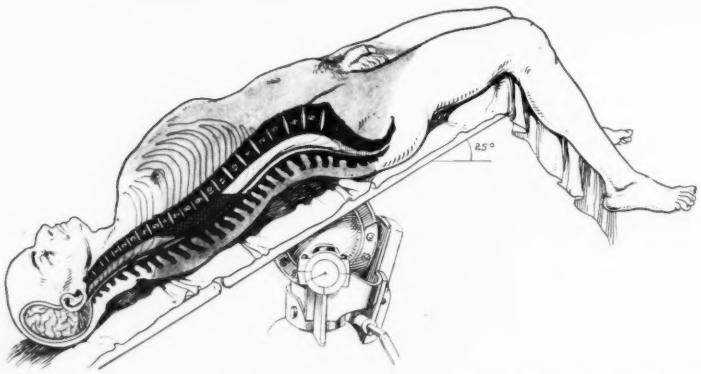


Fig. 9. The position of the anæsthetic charge and the area of loss of sensation in high spinal zone anæsthesia.

opposite direction. If fluid appears instead of air on further aspiration, as recognized in the capillary glass tube, a few more cubic centimeters of air are injected until on still further aspiration air appears in the capillary tube. One aspirates until the first drops of fluid are observed in the glass tube. This signifies that the fluid level within the dural sac corresponds with the point of the needle (Fig. 5).

Now 5 more cubic centimeters of air are injected in order to raise the fluid level somewhat higher cranialward. In this way "a shot field" is prepared for the placement of the "anæsthetic charge" in an area free of spinal fluid. The stopcock of the smaller syringe is now turned so as to connect it with the cannula, and the piston of the small barrel is set by the screw so that no more than 2 centimeters of the contents can be expelled. The lateral opening of the cannula is turned cranialward. Now the piston of the small syringe is pressed forward fairly firmly down to the catch, expelling the 2 cubic centimeters of the anæsthetic charge cranialward (Fig. 6). We designate this maneuver by the combative term "head shot." Immediately after, the stopcock is reversed and 10 or 7 cubic centimeters of air are added depending upon whether high abdominal or low abdominal anæsthesia is desired. *At this stage the connection between the large syringe and the dural sac is closed by turning the stopcock of the smaller syringe.* That is very important for otherwise during the ensuing period of waiting the anæsthetic fluid or the air under pressure will flow back into the large cylinder. The fluid contents of the smaller syringe are not affected by it. A three minute hourglass is now set up.

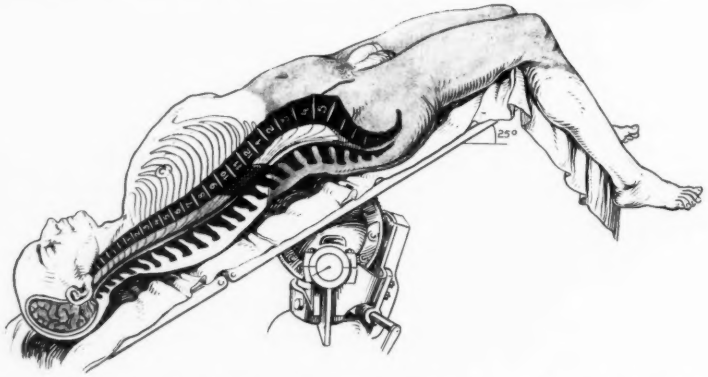


Fig. 10. The position of the anæsthetic charge and the extent of anæsthetic area in low spinal zone anæsthesia.

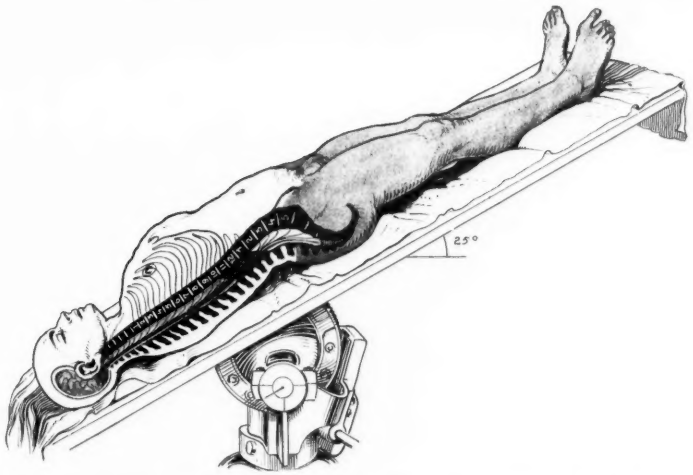


Fig. 11. Position of the anæsthetic charge and spread of anæsthesia in lower extremities anæsthesia.

During the "head shot" the percaine charge is projected cranialward from the puncture needle. It does not! spread cranialward because of its lighter weight!! and because it is not miscible with the spinal fluid the special anæsthetic solution is swimming on the liquor level. The added injections of air send it still farther into the thoracic segment of the spinal cord, thus initiating the paralysis of the corresponding spinal roots (Fig. 7). The roots which lie above or below the anæsthetic charge remain unaffected.

We wait full 3 minutes with the position of the patient and of the apparatus unchanged, at the end of which time the upper limit of skin anæsthesia to pinpricks is tested. If the anæsthesia has not progressed far enough cranialward, which is commonly the case, 5 more cubic centimeters of air are injected after reversal of the stopcock of the small syringe. This has the effect of sending the anæsthetic charge still further cranialward. The stopcock is once more reversed and the hourglass is again set up. The position of the anæsthesia zone is again determined and if necessary, once more regulated by the addition of air.

If the anæsthesia has been found to have reached too far in a cranial direction, as may readily happen at the very first filling in a case of an abnormally narrow spinal sac, then about 5 cubic centimeters of air are removed by turning the stopcock of the air syringe in the opposite direction (Fig. 8). This will cause the anæsthetic charge to sink somewhat toward the buttocks. The stopcock of the small syringe is once more reversed and the zone of anæsthesia again determined after a 3 minute wait.

If at first or after a secondary addition of air, the border of anæsthesia is found to be correct, then no more air is added (Figs. 9 and 10). The greatest amount of air in my experience so far

Fig. 12. Position of the anæsthetic charge and anæsthetic involvement in breech anæsthesia.

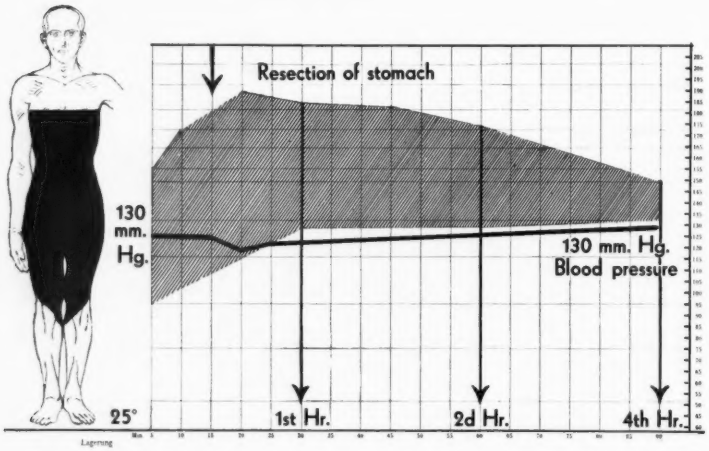


Fig. 13. Time chart and blood pressure curve in high anæsthesia with a completed zone limitation in a case of gastric resection.

injected was 35 cubic centimeters. This amount, however, is rarely required. As a rule 25 to 30 cubic centimeters will suffice. One can wait longer than 3 minutes to determine the exact involvement and that is particularly worth while after what is likely to be the last fill. One should not omit in any case even at the first testing to look for skin anæsthesia high up in the interscapular region. It is easy to overlook this and to add more air erroneously and thus to extend the anæsthesia entirely too far cranialward. Having attained the proper level we now attend to the proper depth of the anæsthesia. If it is found that a pinprick still causes pain we add 0.5 or 1 cubic centimeter of percaine secured from escaping from the rubber tube by sending a little air after it. This can be visually controlled by watching the connecting glass tube. After a wait of 3 minutes the depth of anæsthesia is again tested and if necessary more solution is injected. The anæsthetic dose varies considerably between 2 and 8 cubic centimeters, the average being 3 cubic centimeters. It is advisable to deepen the anæsthesia through the addition of 0.5 cubic centimeter to the required dose if the operation is to last more than 2 hours because the duration of anæsthesia stands in a definite relation to the amount injected.

One should also take into account while giving additional air or drug that extension upward and deepening of the anæsthesia will continue for at least 10 minutes. One should systematically attempt to obtain the lightest degree and the lowest level of anæsthesia compatible with the carrying out of the operation. In every instance, this aim can be accomplished by one trained. In order to save time the expert can make the additional charge of air (5 cubic centimeters) plus the anæsthetic charge (0.5 to 1 cubic centimeter) together so as simultaneously to affect the spread of the anæsthesia upward as well as to deepen it. This maneuver is useful when it is found desirable to advance the zone of anæsthesia somewhat higher or when one feels that the anæsthetic effect is beginning to wear off.

As soon as the proper height and degree have been reached, the needle is removed and the patient is placed in the desired position for the operation without, however, even for an instant changing the position of his body from that of 25 degrees' inclination.

b. *The technique of low spinal anæsthesia.* The low spinal anæsthesia is carried out in a somewhat different manner. The lumbar puncture here is made as low as possible, either between the fourth and the fifth or between the third and fourth lumbar vertebræ. To bring about anæsthesia of the lower extremities, 12 cubic centimeters of cerebrospinal fluid are aspirated. Air is added until on aspiration the fluid level within the sac corresponds to the point of the needle. At this juncture 2 more cubic centimeters of air are added to create room for the charge. With the lateral opening of the cannula turned caudad 2 cubic centimeters of the anæsthetic charge are gently expressed, "pelvic shot." Additional air secures complete expulsion of the charge from the rubber tube (Fig. 11). The stopcock of the small syringe is reversed and anæsthesia is determined after a

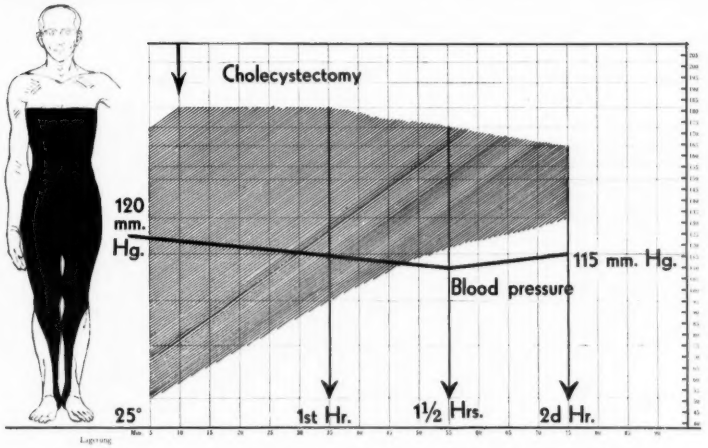


Fig. 14. Time chart and blood pressure curve in high anæsthesia with partial involvement of legs in a case of a cholecystectomy.

minute wait. As a rule further additions of air are here seldom required. On the contrary, anæsthetic solution will be added until deep anæsthesia involves both legs.

To bring about breech anæsthesia, 5 cubic centimeters of cerebrospinal fluid are withdrawn. Three cubic centimeters of air are injected. Here with the lateral opening of the cannula directed caudad, 2 cubic centimeters of the anæsthetic charge are injected as slowly as possible, taking up from one-half to one minute. A forcible injection here, with the point of the needle within the spinal fluid and with a small air bubble, could easily force the injected fluid cranialward and mix it with the spinal fluid. This can be readily shown to take place in a mannikin. With a slow and careful injection, the charge will rise above the layer of cerebrospinal fluid and will collect between the latter and the air bubble; that is, above the cerebrospinal fluid and below the air bubble. To complete its emptying from the rubber tube, a little air is slowly injected until the fluid passes the connecting capillary tube (Fig. 12). After the reversal of the stopcock of the small syringe, 3 minutes are allowed to elapse; further extent and depth of anæsthesia are secured as already described by graded additions of 5 cubic centimeters of air or of 0.5 to 1 cubic centimeter of percaine charge.

As soon as the desired extent and depth have been reached, the needle is disconnected. The puncture hole is sealed with a piece of adhesive plaster. The patient is placed in the desired position for the operation, on his back, his face, or on his side, without, however, raising even for an instant the upper trunk or the head, and without altering the twenty-five degree inclination of the table.

CLINICAL RESULTS

My experience up to date includes 700 cases. Among these there were 500 laparotomies, of which 200 were performed for partial gastric resection and 70 for operations upon the bile tract. Correct technique and proper instruments rule out overdosing or failures. Each failure, to determine the proper dose to secure the required depth of anæsthesia or the desired upward extension is unqualifiedly the fault of the anæsthetist. For in the new method of spinal anæsthesia the anæsthetist is no longer a bungling helper but rather a careful observer, watching the dose and the effect and assuming responsibility for both the depth and the involvement, much as an anæsthetist does in inhalation narcosis.

The anæsthetic charge I employ is lighter than the cerebrospinal fluid and does not mix with it. Because of that it spreads out upon the surface of the cerebrospinal fluid like a layer of oil. The active constituent of the anæsthetic charge is percaine, besides a buffered solution of alcohol and dextrin.[1] Percaine has the advantage of producing a longer anæsthetic effect. Even with smaller doses, loss of pain sufficient for operating purposes lasts, as a rule, over 2 hours, and the return of pain does not set in earlier than 4 to 6 hours later. The duration of anæsthesia depends upon the

[1] Percaine can be secured in America in sterile ampuls from Ciba Company, Inc., New York City.

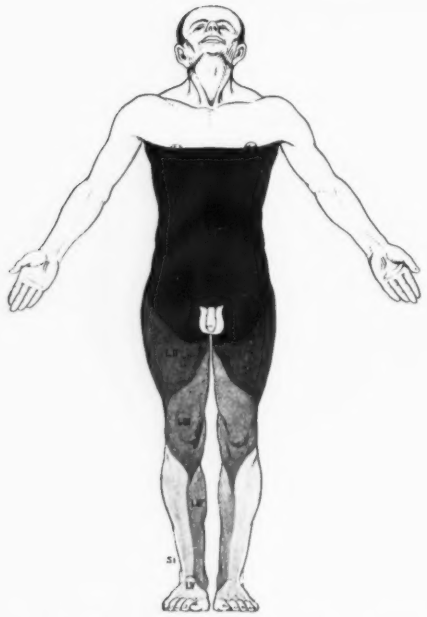


Fig. 15. The depth of the color indicates the duration of the anæsthesia.

concentration of the dose. One gets along with very small doses of percaine because the latter works its effect in a very limited portion of the dural sac and does not mix with the rest of the cerebrospinal fluid. I employ from 2 to 6 cubic centimeters of one-eighth per cent percaine solution, in other words from 2.5 to 7.5 milligrams of percaine. As a rule 2.5 to 3 cubic centimeters of this solution are sufficient. With these minimal doses general toxic manifestations are excluded.

The upper cranial limit of the zone of anæsthesia can be controlled with great accuracy in high spinal anæsthesia. I have deliberately placed it as high as the neck and was able, for example, to operate painlessly in 12 cases of mammary carcinoma with dissection of the axilla. I consider it now as too high, submitting the patient unnecessarily to the danger of respiratory paralysis. I, therefore, deliberately place the upper limit of anæsthesia not higher than the intermammillary line.

The cranial limit in high anæsthesia to begin with is purposely placed rather lower and is only gradually and under the control of cutaneous testing extended upward. In this way the danger of driving the solution too far cranialward in an abnormally small dural sac with resulting respiratory accidents is avoided.

The lower (caudal) limit in abdominal anæsthesia is found in many instances to be in the region of the knees. From here it rises gradually to the symphysis (Fig. 13). Often the area of anæsthesia also involves the calf or the entire limb (Fig. 14). The involvement of the distal roots which takes place here contrary to our theoretical considerations, and in spite of preliminary filling with air, suggests that there is some trickling through of the percaine solution with the resulting temporary paralytic effect, and that the amount of air introduced, in view of the unknown capacity of the dural sac, was too small to begin with to send the charge cranialward from the twelfth dorsal vertebra away from the area of nerves supplying the lower extremities. However, sensation in these cases returns after a relatively short time following the secondary correction of upward placement of the charge as controlled by cutaneous tests (Fig. 14). At the conclusion of the laparotomy the legs are once more sensitive while the abdominal anæsthesia persists for a longer time. The frequent involvement of the extremities in high spinal anæsthesia does not preclude the establishment, after some time, of a strictly limited field of anæsthesia, and, therefore, should be regarded as a *temporary phase* of *zone anæsthesia*. The duration of anæsthesia is represented in Figure 15 by the depth of the color.

When in high spinal anæsthesia the extremities become more or less involved, it appears that the paralysis does not affect the nerves of the blood vessels, at any rate, not completely. The much dreaded blood pressure lowering associated with the older methods is only rarely observed in our procedure (Figs. 13 and 14). A considerable fall of the blood pressure is almost always the result of a technical error, due either to the spread of anæsthesia up to the neck (Fig. 16) or to too big a dose of percaine.

Another advantage not to be underestimated is to be seen in the elevated position of the pelvis which can be at any time increased still further. Cerebral anæmia is thus mechanically combated. The steeper and the longer this elevated position of the pelvis, the more advantageous becomes an otherwise difficult and unpleasant position. The position of the patient is carefully accomplished by degrees and after minutes of waiting. Elevation of pelvis is convenient for most abdominal operations and it is not contra-indicated in operations upon the stomach or liver. The complete relaxation of abdominal walls makes all the viscera readily accessible.

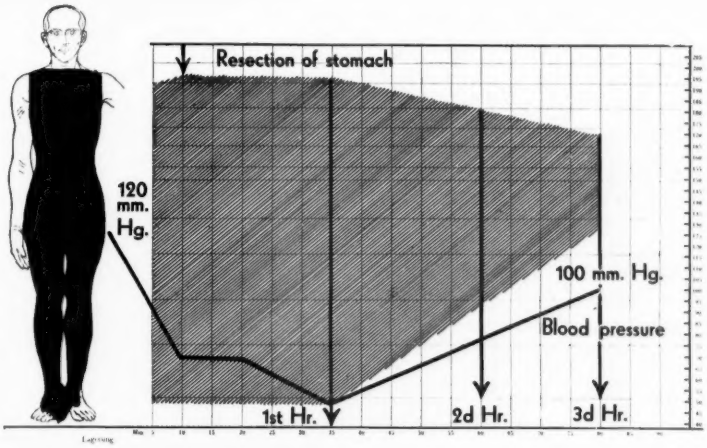


Fig. 16. The time and regional chart, and blood pressure curve in high anæsthesia in a case of stomach resection. The marked fall in blood pressure suggests an error on the part of the anæsthetist; he has driven the anæsthesia above the clavicular line.

Respiratory disturbances may occur as the result of pushing the zone of anæsthesia too high cephalad. As a matter of fact, most patients do not exhibit the slightest sign of respiratory involvement even when the anæsthesia reaches the level of the jaw. By careful aspiration of air the percaine charge can, in cases of inadvertently too high anæsthesia, be lowered back into a safe region. If the condition of the patient appears alarming and the puncture needle has been removed, a new spinal puncture should at once be done and as much of air and percaine solution as possible removed. While this has not so far been necessary, I nevertheless mark the site of the first puncture with a readily recognizable solution and keep on hand an extra sterile needle and syringe. The knowledge of preparedness affords one a pleasant sense of security.

In high spinal anæsthesia, there occur occasionally nausea and vomiting. For this reason I give such patients a suppository of vasano one hour before the operation and when the nausea disappears an ampul of vasano subcutaneously. Ever since we began to adhere to small doses such disturbances have become exceptionally rare, provided, however, that no overdosing takes place and provided the anæsthesia has not been driven too high.

Complicating headaches were exceptional and when they did occur were not severe. Disturbances of ocular muscles or similar late disturbances have not been observed.

The new method of inducing spinal anæsthesia when correctly carried out appears to be as safe as any method can be, local anæsthesia alone being a rival in this respect. For this reason I use this method of inducing spinal anæsthesia systematically in all cases in which it is necessary to operate below the xiphoid process and in which local anæsthesia is applicable, and especially in cases in which general narcosis would endanger life; for instance, patients with arteriosclerosis, anæmia, diabetes, jaundice, nephritis, ileus, peritonitis, cachexia, shock, etc.; in other words, precisely in the cases in which the older methods of lumbar anæsthesia and the use of spinocaine were contraindicated.

ANÆSTHESIA OF THE ABDOMINAL CAVITY

With the new method, the anæsthesia can be extended high enough and made sufficiently deep to enable one to operate painlessly upon the thoracic cavity, the brachial plexus, and even upon the neck. I wish, however, to warn against unnecessarily carrying the anæsthesia too far, and never deliberately to go above the intermammillary line. I take advantage of the fact that the depth of anæsthesia can likewise be regulated at will, and keep it deliberately at its lowest limit much as we like to do in inhalation narcosis. Therefore, I do not regard it as a failure when the patient complains of tenderness in the upper end of a high abdominal incision; to the contrary I welcome it. By means of high pressure local

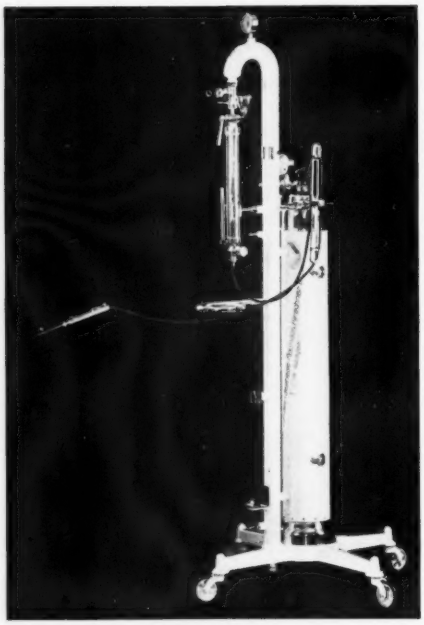


Fig. 17. High pressure local anæsthesia automaton. It ejects the anæsthetic fluid under the pressure of 3.5 atmospheres (Atü).

anæsthesia these sensations are made to vanish in a few minutes. For I am more concerned with the preservation of the patient's strength than with the principle of a rigid and theoretical adherence to one method. Those who prefer a complete anæsthesia can obtain it though without difficulty by increasing the dose.

In operations within the upper abdomen, particularly in operations upon the stomach and the biliary tracts, we enter the domain which is controlled, in addition to the spinal nerves, by the sympathetic and the vagus, and it is the influence of these nerves which renders unpleasant the pull upon the tissues within the upper abdomen. These sensations likewise can be eliminated by deepening the spinal anæsthesia or by advancing it further upward. All forms of anæsthesia, however, including inhalation narcosis, become damaging and dangerous the moment they pass beyond the limit of initial tolerance (cadaver anæsthesias!). I, therefore, advise against pushing this new method to the point which would enable one to exact traction upon the tissues in the upper abdomen without pain. One should abandon this idea but systematically and without waiting for manifestations of pain on the part of the patient, should seek to anæsthetize the sympathetic and the vagus in some different manner. This, in fact, can be accomplished in from 1 to 3 minutes with the help of my high pressure local anæsthesia automaton (Fig. 17). Under a pressure of 3.5 atmospheres (Atü), one-half per cent novocain solution with ¼:1000 percaine-suprarenin solution is injected into the tissues in which these nerves run, in the following manner: After opening of the peritoneal cavity and introducing a retractor, the transverse colon is pulled up and a deposit of anæsthetic fluid the size of a fist is made to rise in the retroperitoneal tissues by injecting into the root of the transverse mesocolon from below upward and in front of the vertebræ. Next, the stomach and the transverse colon are pulled down, a rent is made in the lesser omentum, the caudate lobe of the liver is exposed to view and under the direct control of the eye an anæsthetic deposit the size of a fist is placed below the diaphragm to the left of, and close to, the vena cava (Fig. 18, see first page of article). Finally, a third deposit is placed in the lesser omentum about the stomach walls close to the œsophagus so as to surround the cardia. The entire maneuver consumes hardly three minutes. All tenderness vanishes instantaneously and one may at ease proceed with, say, a gastric resection.

The technique of high pressure local anæsthesia was described by me in earlier communications.

TECHNICAL ERRORS IN THE NEW METHOD OF SPINAL ANÆSTHESIA

A variety of technical errors is possible for the reason that the new method of inducing spinal anæsthesia is not a simple procedure consisting of a single injection of a theoretically computed dose. On the contrary, as in the inhalation narcosis it is a responsible task which, on the part of the anæsthetist, calls for a special knowledge, experience, personal trial, and mastery of technique. The technical errors which cause failures are as follows:

1. Boiling the syringe, tube, and the needle in soda instead of in distilled water. This reduces the effect of the anæsthetic.

2. Failure to maintain a water-tight apparatus, so that air or fluid may escape. This may occur, for example, if the joints of the syringe or its connections have not been oiled. The entire apparatus should frequently be tested under high pressure under water to make sure that it is water-tight. When additional injection of air fails to send the charge cranialward, the apparatus should be examined for leakage.

3. Failure to maintain the patient in the proper inclination of not less than 25 degrees or temporary interruption of position. Disturbing patient will dislodge the air bubble from its caudal position and with it will carry along the anæsthetic solution cranialward.

4. Improper handling of stopcocks of the double syringe during aspiration of the cerebrospinal fluid, during its emptying, or at the time of aspiration of air into the large barrel at the time of injection of air or of the anæsthetic solution into the dural sac, or at the time of its aspiration.

5. Erroneous estimate of air or of the anæsthetic solution to be injected or carelessness in permitting the escape of it from the dural sac.

6. Failure to have the liquor level correspond to the point of the needle at the time of the injection of the anæsthetic charge.

7. Failure to turn the lateral opening of the puncture needle caudalward while injecting air; or not to turn it cranialward while injecting the anæsthetic for high spinal anæsthesia, or again to turn it caudalward for low anæsthesia.

8. Failure to place the "shot" with the customary pressure.

9. Failure to inject air or failure to inject enough of it to complete the expulsion of the anæsthetic solution from the rubber tube.

10. Failure to shut off the air barrel during the waiting interval by leaving the stopcock of the small syringe open, thus permitting the passage of air or of the anæsthetic solution back from the dural sac into the air syringe.

11. Failure correctly to establish the area and the depth of skin anæsthesia, leading to insufficient or to excessive air filling or removal, or to insufficient or excessive injection of additional doses of anæsthesia.

12. Failure to wait full 3 minutes before resorting to additional filling resulting in an overdose. This is particularly likely to occur when the anæsthetist late in starting is being hurried by the surgeon. The induction of anæsthesia should begin about half an hour before the operation. A longer period deepens the anæsthesia but it does no harm because the anæsthetic effect lasts a considerable time.

SUMMARY

The essentially new in the method described may be summed up as follows:

1. The anæsthetic solution is no longer injected directly into the cerebrospinal fluid, leaving to chance the question of it mixing and diffusing in the fluid, but on the contrary is injected in the form of a solution which is not miscible with the cerebrospinal fluid. Furthermore, the anæsthetic charge is placed in a selected segment of the dural sac and is retained there. This is accomplished by filling the caudal portion of the sac with air, the anæsthetic solution separating out in a layer because it is lighter than the cerebrospinal fluid. The localization of the anæsthesia is determined on the basis of skin sensibility by adding or diminishing the amount of air.

2. The patient is no longer given a theoretically calculated dose. In each instance the dose is carefully and sparingly graded in accordance with the effectiveness and the resulting spread of the injected solution.

3. The individual dosage and the limiting of the anæsthetic charge to a small area permit of extraordinary reduction in the size of the dose, markedly diminishing the known and the unexpected accidents of former methods.

4. The anæsthetist is no longer a bungling helper whose task is summed up in the injection of a definite dose of an anæsthetic into the spinal canal. He is now called upon to bring into play in each instance, as in inhalation narcosis, individual skill in localizing and grading the anæsthetic charge, basing his decisions beforehand on physiological considerations. Unsatisfactory results or failures are no longer ascribed to bad luck, but are charged up to the anæsthetist. For this reason, the new methods calls for understanding, technical mastery, faultless necessary instruments, and affectionate devotion.

FROM THE NEUROSURGICAL CLINIC, UNIVERSITY HOSPITAL

RESECTION OF PITUITARY ADENOMATA

CHARLES H. FRAZIER, M.D., Sc.D., F.A.C.S., PHILADELPHIA

IN the Neurosurgical Clinic of the University Hospital, the first operation for exposure of a pituitary lesion was performed, in 1912, by the transfrontal route. Influenced by the experiences of Hirsch, in 1914, I abandoned the transfrontal route in favor of the endonasal approach. The latter approach was employed almost exclusively from 1914 to 1925, when I returned to the transfrontal method.

The endonasal method had much to be said in its favor. The mortality was low, the cosmetic result perfect, but recurrences could not be avoided. Obviously by any transphenoidal route one can but remove the intracapsular contents of the tumor. The capsule itself, at least the major portion of it and all of it in contact with the optic nerves and chiasm, remains intact. Hence, anything approaching a radical procedure was out of the question.

Since 1925, when I resumed the transfrontal approach, I have modified the technique from time to time and, claiming no originality for any individual step, I will describe the procedure as now practiced in my clinic. That the risks of operation have been reduced measurably in recent years is well known. With the technique now employed I had a series of 36 consecutive cases with but 1 death, when coincident with one of those unexplained invasions of streptococcic infections in the clinic, the thirty-seventh case died of meningitis a week after the operation.

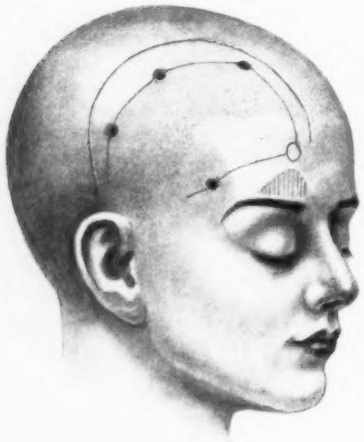


Fig. 1. The incision in the scalp does not follow the outline of the bone flap.

THE OPERATION

Anæsthesia. While avertin anæsthesia is employed now in many of our cranial explorations, I prefer local anæsthesia for pituitary operations. The operation can be conducted painlessly except when separating the capsule of the tumor from the anterior wall of the sella turcica. At this point the patient may complain acutely of pain and this may be relieved by an injection of a 2 per cent novocain solution into the capsular wall. Whether to approach from the right or the left side will depend upon the degree of optic atrophy in each disc. Usually atrophy is much farther advanced on one side than on the other. In fact, vision may be wholly lost in one eye. When there is marked asymmetry I approach from the side on which vision is the more acute. Other things being equal, I prefer the right to the left approach.

Incision. The incision begins 2 centimeters below the hair line, midfrontal, and curves gradually around to terminate above the ear (Fig. 1). For cosmetic reasons I have gradually shortened the incision. One can see in the illustration (Fig. 2) the three transitional stages. The first incision began in the temple, followed the supra-orbital ridge to the midline, thence upward into the hair line, and down to the temple again, thus fashioning a rectangular flap. Later the limb along the supra-orbital ridge was abandoned and the incision began at the root of the nose and thence upward. And now the incision starts just a short distance below the hairline—most of it is concealed within the hairline and eventually is practically invisible (Fig. 3).

Fig. 2. Representing the three transitional stages from left to right.

Fig. 3. The patient 16 months after operation. Scar invisible.

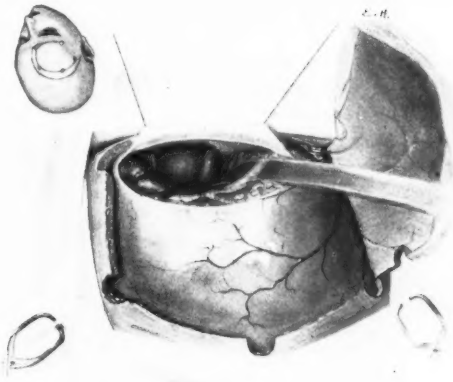


Fig. 5. The approach to the sella turcica is from the side following the direction of the greater wing of the sphenoid bone. One sees presenting the tumor and its relation to the optic nerves.

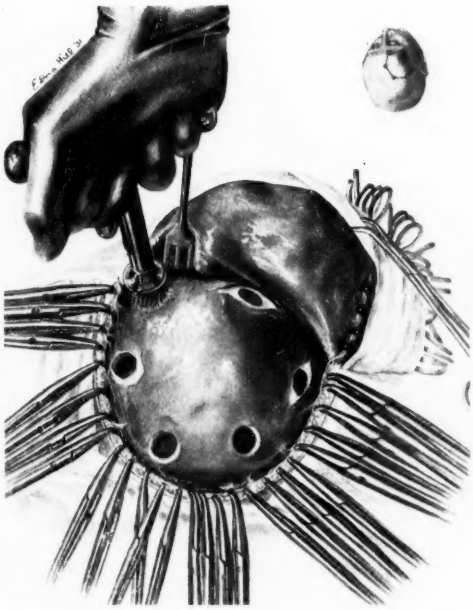


Fig. 4. In the "dual" flap operation, the scalp flap is reflected forward; the bone flap will be reflected temporalward.

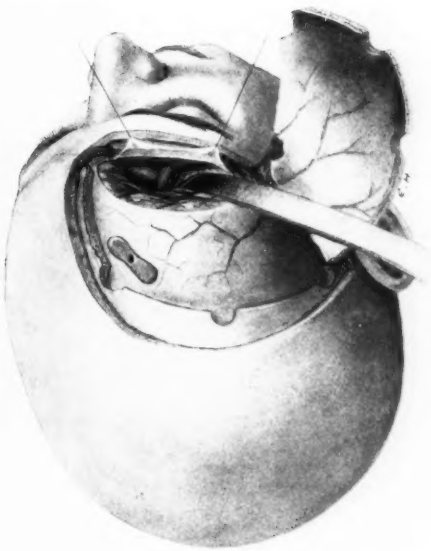


Fig. 6. The flanged cannula introduced into the anterior horn of ventricle to relieve pressure. The scalp flap has been removed to make clearer structures in pituitary fossa.

Fig. 7. Displacement of optic nerves varies with size of tumor. Section of tumor being removed with electrical wire.

The flap. I think it was Souttar who first called my attention to the futility of leaving the scalp attached to the underlying bone in osteoplastic resections of the skull. It was then that I adopted what might be called the "dual" flap technique; as the term implies, there are two flaps, one the scalp the other the bone. In this operation the scalp flap is reflected forward (Fig. 4), and the bone flap with temporal muscle attached temporalward.

In fashioning the bone flap the anterior limb should be as near the base of the skull as possible. The nearer the base of the skull it is, the less will the frontal lobe have to be elevated. But one must be guided by the size of the frontal sinus and in acromegalics often the sinus is of unusually large dimensions. The first perforation is made with a conical trephine, a button of bone removed to be replaced after the operation. This is the only perforation not within the hairline or beneath the temporal muscle. If this perforation were not repaired with the button of bone there would be a visible depression in the middle of the forehead. Usually four more perforations are made. The superior margin of the flap is parallel and 3 centimeters from the midline. The base of the flap corresponds to a line projected from the external canthus of the eye. If the base of the flap were lower than this the middle meningeal artery might be torn as it traverses the groove in the anterior inferior angle of the parietal bone. Thus, what often proves to be a troublesome source of hæmorrhage is avoided.

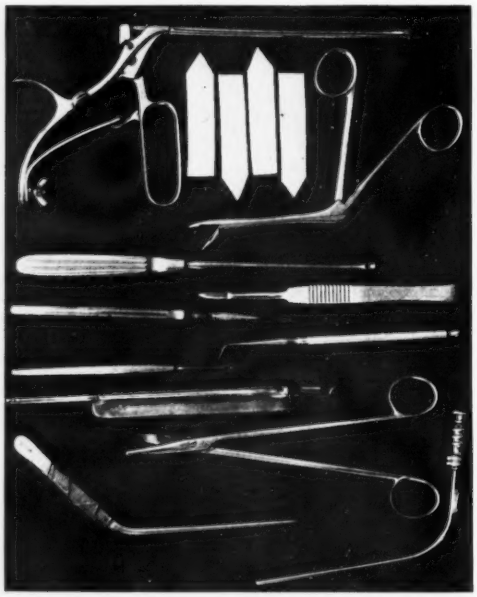


Fig. 10. Special instruments employed in pituitary operations.

The dura. My approach to the sella turcica is intradural not extradural. Once the flap is reflected an incision is made in the dura parallel with the anterior margin of the cranial defect with a slight curve forward into the perforation at either end. Sutures are introduced in the anterior margin of the dural incision for traction purposes. The exposed surface of the dura is protected with a cotton square.

The approach. We come now to what I regard as the most important feature of this operation, namely, the direction by which one approaches the sella turcica. Originally, choosing the shortest course from the cranial wall to the sella, I approached almost directly from before backward. In previous communications I have indicated why this course was objectionable. Today the approach to the sella turcica follows strictly the margin of the greater wing of the sphenoid bone (Fig. 5). Now, in some instances, it may be that the anterior and inferior margins of the cranial opening may not permit of approach on a plane with the greater wing of the sphenoid

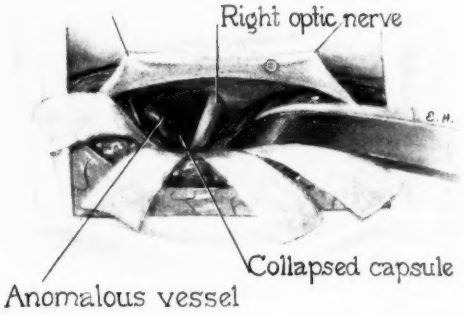


Fig. 8. Capsule collapsed after evacuation of cyst.

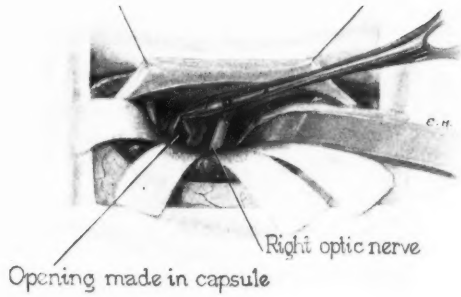
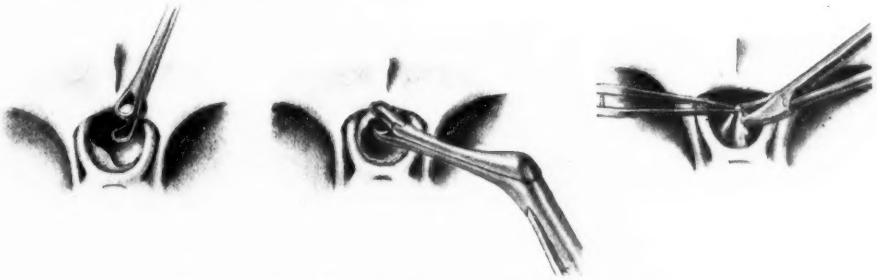


Fig. 9. The initial capsular incision.



Figs. 11 and 12. Resection of capsule with special punches.

bone, and under these circumstances I have enlarged the cranial opening with rongeur forceps downward and forward. As this area of bone is beneath the temporal muscles, the resulting defect will not be apparent and will soon be repaired.

Elevation of the frontal lobe. From this point on to the closure of the wound, the room is darkened and the operative field is illuminated with our special brain retractor mounted with incandescent light. Every effort should be made to avoid harmful pressure or traction on the frontal lobe and especially the region of the tuber cinereum. Most pituitary adenomata are within the confines of the sella turcica and there is no increase in intracranial pressure. Occasionally the tumor may protrude sufficiently above the plane of the sella to impinge on the third ventricle. A ventricular block is thus established and intracranial pressure is measurably increased. One cannot explore the sella turcica safely under conditions of increased pressure. Hence one must tap the anterior horn of the ventricle before attempting to elevate the frontal lobe (Fig. 6).

One should proceed cautiously, the surface of the brain should be protected with paraffin tapes as one advances slowly, centimeter by centimeter. The cerebrospinal fluid as it wells up from the basal cistern is evacuated with the suction cannula. There always seems to me to be an excessive amount of cerebrospinal fluid present with pituitary adenomata, and the more fluid one can remove the greater the ease with which the frontal lobe may be elevated; in fact in some cases the frontal lobe recedes sufficiently by gravity to give one ample exposure for the subsequent maneuvers.

The first landmark to be seen is the olfactory nerve, right or left, as the case may be and, just

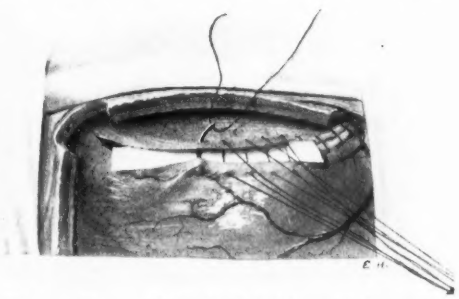


Fig. 13. Closure of the dural incision with interrupted silk sutures.

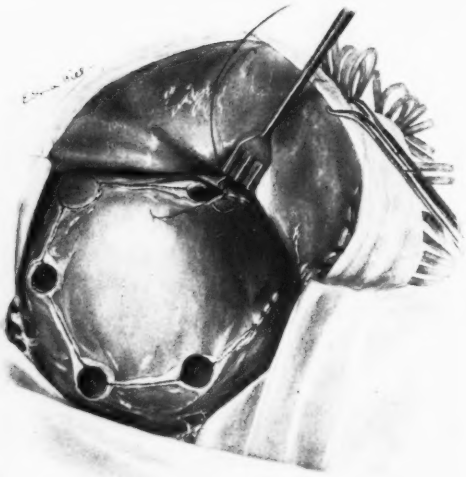


Fig. 14. Flap replaced, button of bone returned to perforation below hair line, closure of incision in temporal muscle.

beyond, the right or left optic nerve. In this "sphenoidal wing" approach to the sella turcica the several veins which pass from the tip of the frontal lobe to the falx are not disturbed.

The intrasellar maneuvers. Once the optic nerve is seen it will soon be apparent whether we are dealing with an operable or inoperable lesion. Fortunately, the large majority of pituitary adenomata are prechiasmal. (I never have been able to secure a satisfactory exposure of a retrochiasmal lesion.)

The capsule of the adenoma presents between the optic nerves and in front of the chiasm. The length, direction, and confirmation of the nerves will depend upon the size of the tumor (Fig. 7). The larger the tumor, the longer the section of nerve from optic foramen to chiasm. This distance may be from 1 to 3 centimeters—the broader the tumor the greater the displacement of the nerves outward—so that their course from foramen to chiasm is not direct but describes an arc with its convexity outward. The longer the duration of the lesion the more will the nerves be flattened, ribbon-like, because of constant pressure.

Aspiration. A bluish discoloration of the capsule usually signifies a cyst. At all events routinely an exploring needle with syringe attached is introduced into the tumor and fluid, if present, is aspirated. This often, though not always, depending upon the flexibility of the capsule, will relieve tension (Fig. 8).

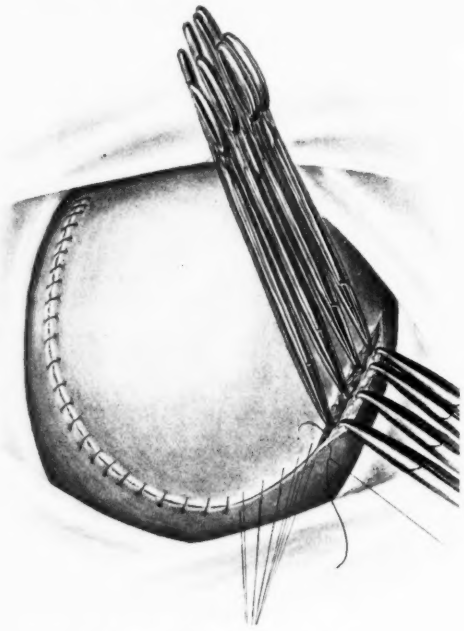


Fig. 15. Closure of scalp incision.

Capsular incision. With a sharp pointed bistoury an incision is made in the capsule (Fig. 9). With a small curette introduced through this incision the glandular contents are removed, fragment by fragment. A specimen may be sent to the laboratory for immediate diagnosis, although from the naked eye appearance one can identify the tissue as that of an adenoma.

Liberation of capsule. With my dural separator the capsule is separated, with the greatest gentleness, from the optic nerves on both sides, the chiasm behind, and the anterior wall of the fossa in front. (At this time and only at this time, the operation being under local anæsthesia, will the patient complain of pain.) I emphasize the importance of gentleness because the optic nerves or chiasm, if damaged, cannot repair themselves and what may seem a very trivial insult may result in permanent impairment of vision.

Capsular resection. There remains now to resect that portion of the capsule which has been mobilized (Figs. 10, 11, 12). The floor of the capsule is left intact. This step of the operation, obviously the most important, requires patience and ingenuity. One is working at some distance from the surface, the cavity of the sella turcica is not large,

and on either side and behind are the optic nerves and chiasm. One must avoid undue pressure or traction upon these at all costs. The capsule is often exceedingly vascular, and one must stop from time to time to control bleeding from its margins. In the process of resection one may use a long handled, narrow bladed pair of scissors or a special capsular punch one or both. Hæmorrhage from the margin of the capsular incision is controlled with silver clips.

Hæmostasis. Perfect hæmostasis is desirable. If there be any oozing from the remnant of capsule or the floor of the sella, pledgets of cotton saturated with adrenalin solution 1:1000 may be used or occasionally a tiny muscle graft.

Now that the intracranial maneuvers are concluded I have no objection to light ether anæsthesia. The patient has been under considerable restraint and tension. He welcomes an opportunity for relaxation. Light ether anæsthesia is most welcome and under its influence the wound is closed, first the dural incision with interrupted silk sutures (Fig. 13), then the wounds in the temporal muscle and aponeurosis (Fig. 14), and finally the scalp (Fig. 15). In closing the dural incision, I find it an excellent plan, as a means of protecting the cortex, to place a paraffin tape beneath the suture line. After the bone flap is replaced, the button of bone is inserted in the perforation from which it was removed. A counter opening is made for a rubber tube inserted between scalp and cranium. I have found it desirable to drain this space for 24 hours.

In operations for the removal of pituitary adenomata, there is striking uniformity in the physical condition with which the operator is confronted. The location of the tumor never varies; its relation to the adjacent anatomical structures is always the same. Hence it is possible to standardize one's technique, and the operation described may be applied to every case without variation.

TRAUMATIC SEPARATION OF THE SYMPHYSIS PUBIS DURING SPONTANEOUS LABOR

With a Clinical and X-ray Study of the Normal Symphysis Pubis During Pregnancy and the Puerperium[1]

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RUPTURE of the symphysis pubis during spontaneous labor and associated with clinical symptoms is rare. This occurrence, observed in 5 patients during a period of 5 years, led to an investigation of the entire subject.

Search of the world's literature yielded a total of only 62 instances of rupture of the symphysis pubis following spontaneous labor. Seventeen of these were reported by Kehrer in 1915. He listed 101 ruptures of the symphysis pubis, the remaining 84 having followed forceps or breech extraction. This approximate ratio of one rupture following spontaneous labor to five following operative deliveries no longer exists since high forceps delivery has been largely replaced by cæsarean section. Twenty additional case reports of rupture of the symphysis pubis following spontaneous labor were found antedating 1915, and an additional 25 from 1915 to date.

This total of 67 instances of rupture of the symphysis pubis with clinical symptoms following spontaneous labor—including the 5 reported here—range in frequency from 1 in 5,000 deliveries to 1 in 30,000. The 5 cases reported here occurred among the last 25,000 deliveries at the Michael Reese Hospital from 1912 to 1931. Nemec reports an incidence of 1 in 6,072, von Fernwald 1 in 10,000, Schauta 3 in 30,000, Sauer 3 in 64,000, and Kayser found only 3 instances in 94,000 consecutive deliveries in the Schauta and Chrobak clinics.

In an X-ray study of the symphysis pubis in 54 consecutive patients during pregnancy, labor, and the puerperium, Brehm and Weirauk reported the finding of severe separation with symptoms (a separation of more than 0.9 centimeter) in 26 per cent of the series. They further report a slight separation without symptoms in 27 per cent (0.5 to 0.9 centimeter). Only 47 per cent of their series showed no separation. They concluded that separation occurs more frequently than formerly supposed, that separations of less than 1 centimeter will not produce symptoms and that separations of more than 1 centimeter will produce the symptoms typical of traumatic separation. A frequency of 26 per cent of separation of the symphysis pubis with symptoms and corroborative X-ray evidence is quite at variance with this or any other article in the literature. Moreover, X-ray findings are useless as a diagnostic aid in this type of injury unless the rupture produces a gross separation.

It is well known that injury to the ligaments of the symphysis pubis can and do occur without the production of any clinical symptoms. Gmelin found a 1.5 centimeter gap while doing a cæsarean section, Ahlfeld found a marked separation in a patient who died in labor, and Zulauf and Loeschcke found marked separations of the symphysis pubis repeatedly at the autopsy table.

ANATOMY OF THE SYMPHYSIS PUBIS

Cunningham describes the symphysis pubis as being an amphiarthrosis. The two pubic bones are covered with hyaline cartilage with interposed fibrocartilage in the interior of which there is a vertical anteroposterior cleft. This cavity appears at about the tenth year and results from the breaking down of the interpubic lamina, there being no synovial stratum. The joint is reinforced by four ligaments, the superior and posterior ones being very weak and consisting of scattered fibers passing between the two bones. The anterior pubic ligament is thick and strong, the fibers are oblique and form an interlacing decussation. The arcuate pubic ligament occupies the arch of the pubis, is of considerable strength, and gives roundness to the pubic arch. It is quite thick, is attached anteriorly and laterally to the bones, but is free in its inferior border.

ALTERATIONS OF THE SYMPHYSIS PUBIS IN PREGNANCY

Pregnancy produces definite changes in the ligaments of the symphysis pubis as well as in those which reenforce the sacro-iliac synchondroses. This has been repeatedly confirmed experimentally and by autopsy findings. From the first mention of these changes by Hippocrates to the present time, innumerable writers have found an increased mobility in the joints of the pelvic girdle

[1] This work was aided by a grant from the Rosalinda Klein fund of the Nelson Morris Institute for Medical Research. Read before the Chicago Gynecological Society, June 19, 1931.

due to the increased blood and lymph supply which accompanies pregnancy, thus producing what really amounts to an hypertrophy of the ligaments. These changes have been found early in pregnancy. Both Driver and Muellerheim found definite movement in the pubic joint in pregnancy. Fernwald found a 3 to 5 millimeter gap in the pubic bones. He found this by placing the examining finger against the lower surface of the symphysis pubis through the anterior vaginal wall, and having an assistant make pressure against the two trochanters and then suddenly release this pressure. Engstroem placed the index finger in the vagina against the symphysis pubis, the thumb against the joint externally and, while the patient shifted the weight of her body from one foot to the other, could determine mobility in the joint. Loeschcke examined the joints of four multiparæ on the autopsy table, all of whom died in the first 24 hours postpartum, and found definite mobility of the symphysis pubis in each case.

ETIOLOGY

Various hypotheses have been advanced to explain rupture of the symphysis pubis. Kehrer states that rupture is due to a pathological exaggeration of the physiological softening and distention of the entire pelvis. In support of this, both Eldridge and Eisenberger report women in whom pubic mobility became more marked through successive pregnancies. Both report that separations occurred in these pregnancies which became more marked throughout the successive pregnancies. Gusserow has reported the case of a multipara in whom all three pelvic joints became markedly relaxed each time she became pregnant. Each successive pregnancy resulted in such an increase in this mobility that, with the third and fourth pregnancies, she found herself unable to walk or stand. This relaxation disappeared immediately following delivery each time, and between pregnancies, the pelvic joints were apparently normal and symptomless. Such an explanation, however, fails to take into consideration the mechanics involved in true rupture of the symphysis pubis and this type of lesion should be regarded rather as a relaxation of the involved joints.

Lehman, Bardeleben, and others feel that rupture is due to a lack of flexibility together with a distention of the pelvis. They believe that rupture occurs only when the pubic joint does not give, i.e., when there is pathology present in the joint. Such pathology may be due to a congenital hypoplasia, caries, osteomalacia, arthritis, rickets, or contracted pelvis. This group of causes includes two of the factors which may play an important causative rôle, i.e., bony deformity or undervelopment of the connective tissue structures. Boddaert found osteomalacia in his patient; Glenn and Colwell each found tuberculosis of the symphysis pubis; Montanelli's patient was rachitic and the patient reported by Jellinghaus had suffered from a polyarthritis for years. Holzbach reports 3 patients all of whom gave definite evidences of generalized hypoplasia of all the connective tissue structures. This was also found in 2 of the patients reported in this series.

That such a hypoplasia is not an essential factor is demonstrated by the occurrence of rupture in the patient reported by Keller. This patient did not give evidence of a generalized underdevelopment. On the contrary, she was a large muscular woman who had spent some years as a gymnasium instructor. Keller's explanation seems by far the most logical. He states that *rupture of the symphysis pubis in spontaneous labor is due to marked intensity of the uterine contractions plus marked rapidity of labor.* The validity of this explanation is completely corroborated by the analysis of this entire series.

In this series, 14 women were under 25 years of age, 31 were between the ages of 25 years and 35 years, and 12 were over 35 years. The average age of the entire group was 29 years. These figures would not seem to be significant and age, therefore, apparently plays no rôle.

There were 18 primiparæ and 46 multiparæ. Of the latter, 13 were in their second pregnancy, 19 in their third, 6 in their fourth, and 2 in their fifth pregnancy. Six had had more than seven pregnancies. The 46 multiparæ represent 73 per cent of the total number in whom parity was mentioned. This preponderance of multiparæ in whom "marked intensity of uterine contractions and marked rapidity of labor" is much more apt to occur than among primiparæ bears out Keller's explanation as to the etiology of separation of the symphysis pubis.

The type of previous labors is apparently of no significance. Seven women had had short or easy labors, 11 had had normal labors, 4 had had long hard labors, and 3 had been delivered by forceps.

Pathological pelves were found in 15 patients, 12 being characterized as contracted, 2 as flat rachitic, and 1 as osteomalacic; 24 were classified as being normal. This gives an incidence of 39 per cent for contracted pelves.

There were 33 babies overweight, 22 weighing 7½ to 10 pounds, 7 weighing over 10 pounds, and 4 being classified as very large. This gives an overweight incidence of 67 per cent. The dis-

proportion due to a contracted pelvis may be identical with the disproportion produced by an oversized fetus. Here is the background for the violent or fulminating type of uterine contractions which is one of the causative factors in the production of separation of the symphysis pubis.

Labor was characterized as short in 6 instances. The length of labor was given as less than 1 hour in 6, 1 to 4 hours in 8, 4 to 12 hours in 24, and over 12 hours in 14. In other words, in 20 out of 58 (34 per cent), labor was unusually short.

In 5 instances delivery occurred precipitately after a particularly violent pain, 16 labors were classified as fulminating, 7 as easy, and 9 as hard; 21 out of the 37 reporting on the type of labor, an incidence of 57 per cent, were therefore described as having "marked intensity of uterine contractions."

MECHANICS

The force necessary to tear the pubic ligaments and permit rupture of the fibrocartilaginous symphysis pubis has been determined experimentally. Poulett, in 1864, used 7 female pelves and found that it required between 170 to 200 kilograms of direct pull to rupture the symphysis pubis. Thirty years later Fessler repeated these experiments and corroborated the findings. In addition he took a block of wood, fashioned it so that it would exactly fill a female pelvis, and then soaked the pelvis and wood in water. The expanding wood exerted enough pressure to rupture the symphysis. Obviously the mechanical problem involved in separation of the symphysis pubis is duplicated by the expansion test discussed by Fessler rather than by the direct pulling asunder employed by Poullet.

The force that causes rupture is a wedge effect produced by the violent thrust of the fetal head through the superior strait under the combined power of the uterine and voluntary musculature. This must be true since the maximum contractile power of the uterus is something under 25 kilograms and the estimated additional power of the voluntary musculature is another 25 kilograms (Schatz). This total maximum of 50 kilograms is approximately one-quarter of the force necessary to rupture the symphysis pubis, when applied by a direct pull.

The question of involvement of the sacro-iliac joints was tested by Wishner and Mayer. They took a female pelvis, cut through the symphysis, and pried the pubic bones 4 centimeters apart. This resulted in the anterior sacro-iliac ligaments tearing and giving away so that the right joint gaped 1 centimeter and the left one 0.5 centimeter. It should be noted that, in this work, damage to the sacro-iliac joints required the production of a 4 centimeter gap between the pubic bones, a lesion which practically never happens in spontaneous labor, although it is conceivable after operative delivery. Opinions as to involvements of the sacro-iliac joints differ diametrically. Ahlfeld and Wishner and Mayer state that they must be involved in true rupture of the symphysis pubis while Kayser, Muellerheim, Zweifel, v.Fernwald, and Engstroem state that such involvement of the sacro-iliac joints is not necessary.

When the sacro-iliac synchondroses are involved, the torn ligaments are those on the anterior surface of the joints, which accounts for the absence of symptoms other than tenderness referable to these joints (Wishner and Mayer). The sacro-iliac involvement seems to be more common on the right side. In analyzing the literature, sacro-iliac joint involvement was mentioned 22 times. Both joints were involved in 6 patients, the right joint alone in 8 patients, and neither joint was involved in 8 patients. In only one instance was the left joint involved alone.

PATHOLOGY

The nature of the injury is primarily a rupture of the pubic ligaments, after which the fibrocartilaginous union at the symphysis is torn. The bony gap demonstrable by X-ray or by actual palpation is never a criterion of the existence or degree of the injury. Neither is it related to the presence or severity of the clinical symptoms and findings. In 4 of the 5 cases observed by us, the X-ray findings revealed no abnormal separation although all 5 women presented the typical picture of rupture of the symphysis pubis. Eisenberger reports a similar experience. His patient presented all the clinical evidences of rupture, but a widened gap between the pubic bones could neither be felt or demonstrated by X-ray examination. In a report by Mandruzzato, the diagnosis of rupture in 5 patients was based essentially on X-ray findings. As there were no typical symptoms and all of the patients were discharged as cured in a comparatively very short time, they were regarded as unproved and were therefore not included in this report.

There is rarely a complete separation of the joint, a bridge of fibrocartilage nearly always remaining. The separation may, however, be complete. Holzbach reports such a complete separation found at postmortem in a patient who died shortly after delivery. In this patient all of the cartilage had been completely torn and there was no connection present between the pubic bones.

Hæmorrhage into the involved area is usual and results in the accompanying œdema. This extravasation of blood fills the cavities produced by the tearing and separating of the fibers of the ligaments. Such a hæmorrhage may be large or even fatal. Holzbach reports such a fatality in the patient with the complete separation just described. A hæmatoma developed immediately after delivery which increased in size and dissected its way upward not only into the cavum Retzii but also along the anterior belly wall. Continuing hæmorrhage resulted in death 3 hours after delivery.

Infection of the involved area occurs usually when there is a communication into the vagina through lacerations. This may result in abscess formation and such a complication adds markedly to the gravity of the injury and may terminate fatally. Glenn and Colwell each report such a fatality. Abscess formation was reported 7 times in this series (Glenn, Colwell, Hartwig, Puech, Mayer, Benthin, Naujoks). In a series of 98 instances of separation of the symphysis pubis following operative deliveries, Rudaux reported abscess formation 23 times.

Unrecognizable trauma undoubtedly occurs frequently. Delestre, Glenn and Koestlein reported the finding of pubic separation at the autopsy table. Loeschcke in 5 autopsies done on primiparæ all of whom died within 24 hours after delivery, found hæmorrhage into the symphysis pubis three times with tearing of the ligaments but no separation. The cavities produced by the tearing of the ligaments were filled in each instance by a serosanguineous fluid.

Autopsy studies on the normal symphysis pubis reported by Zulauf, Cruveilhier, and Loeschcke indicate that the gap is greater in multiparæ than in primiparæ. The latter author states that these increased gaps are the direct result of acute birth traumata and that they are constant findings in the multipara. He found no gap present in males or in multiparæ. The series of X-ray observations reported here does not bear this out. Many observations on women made before and after spontaneous delivery in both primiparæ and multiparæ showed no appreciable differences in the size of the gap.

ROENTGEN STUDIES

The inadequancy of the X-ray in the evaluation and diagnosis of separation of the symphysis pubis in our own series led to a comprehensive X-ray study of this joint. One hundred and fifty patients were examined roentgenologically. In order to determine a normal standard, a preliminary study was made which included 20 males, 30 non-pregnant females, and 20 pregnant females. The ages ranged from 17 to 43 years. Sixteen per cent of the females were multiparæ. The pregnant women were all from the prenatal clinic of the Michael Reese Hospital. No individual was used for this study who gave a history of injury of either the pubic joint or the pelvis.

A second series, aimed at the X-ray determination and time of occurrence in pregnancy of the known physiological changes in the joint, included 80 pregnant women who were followed roentgenologically from the second or third month of pregnancy up to the time of delivery and again 10 days postpartum. Films were taken in three positions and retaken at 6 to 8 week intervals.

Position. Three positions for projecting the symphysis pubis were used in this study: (1) dorsal position, with the subject on the back, Figure 4; (2) ventral position, with the subject on the abdomen, Figure 5; (3) sitting position, with the subject in a semi-sitting position, with the back at an angle of 45 degrees and the buttocks pressed firmly against an inclined support, Figure 6, in order to place the plane of the pelvic inlet parallel to the film.

The symmetry in each instance was obtained by proper position and was maintained, together with a fixation of the lower extremities, by means of sandbags. The positions varied in the amount of discomfort to the patient in direct relationship to the month of pregnancy, the greatest discomfort always being found in position 2 and just before term. Position 3, the semi-sitting position, was comfortable at every stage of pregnancy and therefore most agreeable to all patients.

It was attempted in the preliminary work to project the symphysis pubis with patient in the upright or standing posture. This position was found to be impractical because of the progressive difficulty of projecting the symphysis pubis due to the protruding abdomen of late pregnancy. No distortion of the symphysis pubis could be found when the body weight was carried on either the right or the left leg. Traction maintained on either leg with patient in the semi-sitting posture also revealed no distortion of the joint, neither was there distortion when traction was maintained on one leg while a direct upward push was maintained on the other leg.

TECHNIQUE

All of the roentgenograms were taken on a Bucky grid. Eight by 10 films were used throughout. The symphysis was localized in each instance with a dental cone 7 centimeters in diameter. A

TABLE I.—CHRONOLOGICAL SUMMARY OF SIXTY-TWO CASES

Author	Date	Age	Para	Previous History	Pelvis	Length of Labor	Type of Labor	Size Baby	Onset
1. Nicholson	1824	21	ii						3 day
2. Riecke	1831	23	iii			24 hr.	Hard	Large	At once
3. Meissner	1833				Contracted	Long	Hard		2d stage
4. Hoffman	1847		ii		Contracted	20 hr.	Hard-face presentation		
5. Hoffman	1847		ii		Contracted				
6. Boddaert	1853			Osteomalacia	Contracted		Hard		Labor
7. Grenser	1853		ii						Labor
8. Arendt	1858	23	ii		Normal	6 hr.	Easy	8 lb.	20 day
9. Fraisse	1858	26	ii	Same in 1st pregnancy					6th. mo. pregnancy
10. Ahlfeld	1876	32	iii		Normal	36 hr.			
11. Ahlfeld	1876	24	i		Normal	15 hr.			1 day
12. Bauteron	1878	35	vii			Short	Easy		
13. Bauteron	1878	24	i			Short	Easy		
14. Eldridge	1884		ii	Same in 1st pregnancy		7 hr.		10½ lb.	8th mo. pregnancy
15. Mangiagalli	1887	36	vii	6 spontaneous labors	Normal	12 hr.	Easy	Twins small	2d stage 2d baby
16. Koestlein	1888	36	viii		Contracted	20 hr.		3,470 grams	
17. Remy	1890		iii					Large	8th mo.
18. Hartwig	1893		iii	1 forceps—1 spontaneous		6 hr.		Large	
19. Glenn	1893	28	iv			6½ hr.	Easy	9¼ lb.	1 day
20. Ludlam	1894	37	iii	2 spontaneous		Short	Easy	14½ lb.	1 day
21. Betz	1894		iii						Labor
22. DeLee	1898	24	iii		Contracted	Very short	Easy	Less than 7 lb.	1 hour
23. Peuch	1899	23	i		Normal	36 hr.	Hard—1 sudden pain then delivery	Average	1 day
24. Jellinghaus	1899	31	vii	All long labors polyarthritis	Normal	6 hr.		4,700 grams	Labor
25. Himmelsbach	1900	25	i			7 hr.	Long hard 2d stage		1 day
26. Colwell	1901	30	iv			16 hr.			
27. Schenck	1902	31	iii			Long	Long hard 2d stage	10 lb.	1 day
28. Bochenski	1904	33	iv	All spontaneous	Normal	1 labor pain	Precipitate on street		Immediately
29. Eastman	1906	27	ii	Short labor		1 hr. fol. by	1 severe pain—delivery	9 lb.	At once
30. Mayer	1906	21	i		Normal	12 hr.		7½ lb.	10 hr.
31. Engstroem	1907		ii						8th day
32. Delestre	1908	19	i		Contracted	76 hr.	Severe	2,870 grams	
33. Montanelli	1908	36	iii	2 long spontaneous deliveries	Flat rachitic	1 hr. 20 mins.	Severe, short	6,000 grams	At once
34. Meyer	1910	36	iii						3d stage
35. Huxley	1910	36	vii	All short labors		8 hr.			2 days

IN LITERATURE AND AUTHORS' FIVE ADDITIONAL CASES

Initial Symptoms	Findings	Sacro-iliac	Duration	Recovery	Subsequent Labors
Stood up, fell unconscious from pain	Pain, immobility, backache	Both	Short	Rapid	
Pain	Right pubis above and in front of left one				
Sudden, severe pain and descent of head-cracking sound	1 finger gap				
			4 wks.	Prompt	
			5 wks.	Prompt	
Sudden crack	Pelvis larger				
Pain	Typical		3 wks.	Good	
Pain on standing, sitting, walking, and turning	1 finger gap			Good	
Pain—could not walk last 3 mo.	1 finger gap—bones movable	Both			
	1.6 cm. gap	Right		Died of sepsis—27 days	
			2 wks.	Good	
			4 wks.	Well	
			4 wks.	Well	
Pain—could not walk. Felt as if pried apart	1 finger gap—2 finger gap while head was in pelvis		Short	Complete	
Pain—sudden as head went through	Left pubis 2 cm. higher and superimposed on right		8 wks.	Complete	Uneventful
Pain	None until autopsy		46 days	Died of sepsis	
Pain and cracking sound and sensation on walking	Marked mobility and separation	Right			
Pain	Separation—abscess 3 wks. later		6 wks.	Complete	
Pain	Tuberculosis found at autopsy		16 days	Died of tbc. sepsis	
Pain	Wide separation		8 mos.		
Pain and grinding cracking noise heard	Pain, tenderness, waddling		1 yr.		
Pain and inability to cross legs	Tenderness and definite groove	Left	7 wks.	Complete	
Severe pain and cracking noise followed by sudden delivery	Typical followed by prevesical abscess—no gap				
Pain and immobility	Tenderness, 5 cm. gap, right pubis lower than left				
Immobility—snap heard on turning	Tenderness—marked mobility—no gap	Right	5 mos.	Persistent pain on walking	
	Swelling—tuberculosis, sepsis, and death		18 days	Died of tbc. sepsis	
Pain on turning and grinding, could not sit or stand	Tenderness, crunching could be felt on moving, œdema		6 mos.	Operated upon—bones wired together	
	1 finger gap, œdema typical		8 wks.	Good	
Pain and grating—snapping noise heard	Mobility and tenderness				
Pain and immobility	Gap typical followed by abscess		Long-abscess		
Pain and cracking heard on getting out of bed	Marked mobility—1 finger gap		14 mos.	Sutured, complete recovery mobility and gap persisted	3 uneventful
None	Found at autopsy—all but posterior ligaments torn—no gap		1 wk.	Died of sepsis	
Sacral and pubic pain	3 cm. separation—X-ray positive		4 mos.	Good, gap persists	
Severe pain and grinding, tearing sound heard during manual removal	1 finger gap—X-ray positive	Both			
Pain followed sudden turn	Tenderness, œdema, no rotation. Left pubis ¼ in. higher, ¼ in. gap	Neither	6 wks.		

TABLE I.—CHRONOLOGICAL SUMMARY OF SIXTY-TWO CASES IN

Author	Date	Age	Para	Previous History	Pelvis	Length of Labor	Type of Labor	Size Baby	Onset
36. Retzbach	1915	24	iii	1 long, 1 short	Flat rachitic	40 mins.	Fulminating		
37. Benthin	1922	28	iv	All spontaneous				Very small	1 day
38. Anderodias	1923	23	ii			1 hard pain	Cracking sound followed by delivery	4,660 grams	Labor
39. Telfair	1923	26	iv	Normal full term		Very rapid	Strong pains		
40. Holzbach	1925	21	i	Very frail		12 hr.	Strong pains		8 hr.
41. Holzbach	1925	39	i	Very frail	Normal	5 hr.	Rapid—strong pains	5½ lb.	1 wk.
42. Holzbach	1925	25	i	Very frail	Normal	8 hr.	Rapid—strong pains	6 lb.	1 hr.
43. Polgar	1925	27	iii	Normal labors	Normal	Short	2d twin delivered by 1 strong pain	2,300 grams 2,490 grams	5 days
44. Nemec	1926	28	iv	All 3 day labors	Contracted	8 hr.	1 severe pain followed by cracking sound—head went from inlet to perineum by one pain	4,250 grams	Labor
45. Eisenberger	1926	29	ii	Same with 1st baby 4,000 grams		3 hr.	Hard labor	4,200 grams	Labor
46. Heyman	1927	26	i		Normal	14 hr.		4,000 grams	1 day
47. Naujoks	1927	40	v	All easy and short	Normal	10 hr.		3,220 grams	At once
48. Olow	1927	36	i		Contracted	21 hr.		3,720 grams	
49. Busalla	1928	33	i			31 hr.			1 day
50. Denntzer	1928	42	i		Normal			3,000 grams	Labor
51. Denntzer	1928	38	i			3 hr.	Short and hard—crack felt and heard	3,000 grams	Labor
52. Pechnikoff	1928	30	i		Contracted	8 hr.		2,770 grams	1 day
53. Pechnikoff	1928	27	i		Normal	6½ hr.		4,150 grams	3 days
54. Keller	1928	30	iii	Normal deliveries		5½ hr.		3,100 grams	1 day
55. Keller	1928	21	i	Gymnasium teacher		8 hr.	Violent pains	4,020 grams	1 day
56. Keller	1928	27	ii	Forceps delivery	Normal	6 hr.		3,950 grams	1 day
57. Keller	1928	27	iii	Normal deliveries	Normal	4½ hr.	10 mins. 2d stage	3,450 grams	3d day
58. Shordania	1929	31	v	Normal deliveries	Normal	1½ hr.		3,650 grams	1 day
59. Lusena	1929	25	iii	Normal deliveries	Contracted	Very short	Fulminating, 20 mins. 2d stage	3,400 grams	At once
60. Lusena	1929	31	iii		Contracted			3,600 grams	1 day
61. Bazan, Goenaga	1930	28	xi	Normal deliveries		10 hr.			At once
62. Wu	1930	27	iii		Normal	12 hr.	15 mins. 2d stage	3,420 grams	At once
63. Reis, Baer, Arens, & Stewart	1931	30	iii	2 short easy labors small, frail woman	Normal	10 hr.	20 mins. 2d stage	4,700 grams	3 day
64. Reis, Baer, Arens, & Stewart	1931	23	i	Small, frail woman very large husband	Normal	10½ hr.	Average	3,430 grams	6 day
65. Reis, Baer, Arens, & Stewart	1931	25	iii	Easy, short labor	Normal	13 hr.	26 mins. 2d stage	4,170 grams	5 day
66. Reis, Baer, Arens, & Stewart	1931	27	ii	Low forceps	Normal	7 hr.	35 mins. 2d stage	3,800 grams	3 day
67. Reis, Baer, Arens, & Stewart	1931	28	iii	Short, easy labors	Normal	1½ hr.	7 mins. 2d stage	3,500 grams	3 day

LITERATURE AND AUTHORS' FIVE ADDITIONAL CASES (Continued)

Initial Symptoms	Findings	Sacro-iliac	Duration	Recovery	Subsequent Labors
Pain and "tearing apart"	Œdema, outward rotation. 2 cm. gap. X-ray positive, gap "normal"	Both	8 wks.		
Pain and tearing sensation	Tenderness, œdema. 2 finger gap. X-ray positive. Abscess		12 wks.	Good, X-ray showed no gap—some ossification	
Loud cracking—1 long pain—delivery	3 cm. gap, œdema	Right		Good	
	X-ray positive				
	1 finger gap		8 wks.	Good	
	1 finger gap		3 wks.	Good	
Pain	Large hæmatoma dissecting up belly wall. 2 finger gap and cartilage all torn at autopsy	Both	3 hr.	Died of dissecting hæmatoma	
Chills and fever 4 days, then pain	Typical, 1 finger gap. X-ray positive		3½ wks.	Complete, no gap left	
Cracking sound in 2d stage	Typical		8 wks.	Good	
Sudden severe pain in 2d stage	Typical but no gap. X-ray negative		6 wks.	Complete	
Pain	X-ray showed 1.5 cm. gap				
Pain on movement	Typical, 2 cm. gap found. Abscess at operation. X-ray positive		8 wks.	Complete	
Pain	Typical			Rapid	
Pain	Tenderness, œdema. No gap, no rotation				
Pain	Typical, X-ray positive		4 mos.	Complete	
Crack heard and felt	Typical, X-ray positive				
Pain	None for 1 mo., then 1 cm. gap. X-ray showed 1.5 cm. gap		6 mos.	Complete	
Pain and swelling	Typical gap, X-ray positive		4 mos.	Complete	
Pain and swelling	Œdema, X-ray showed wide gap			Complete	
Pain and swelling	Œdema, tenderness. X-ray showed wide gap	Right	4 wks.	Complete	
Pain and swelling	Œdema, tenderness			Complete	
Pain	Œdema. Feel gap. X-ray shows slight separation	Neither	3 wks.	Complete	
Pain	X-ray shows 3 cm. gap after 3 mos.	Right	4 mos.		
Pain	Typical, 1 finger gap. X-ray positive		1 mo.		
Pain	Typical, 1 finger gap. X-ray positive				
Pain	Tenderness and gap. Both bones movable				
Pain	1.5 cm. separation. X-ray positive		3 wks.	Complete, X-ray showed 1 cm. separation	
Pain especially on moving	Typical. X-ray shows no gap, but absorption left ramus	Neither	5 wks.	Complete	1 normal
Pain especially on bedpan	Typical and mobility. No gap. X-ray negative	Neither	4 wks.	Complete	
Pain and pain in right hip	Typical, X-ray positive	Right	5 wks.	Complete	
Pain	Typical, X-ray negative	Neither	3 wks.	Complete	2 normal
Pain—pneumonia at same time	Typical and pneumonia then pleurisy and effusion	Neither	8 wks.	Complete	

constant target distance of 36 inches was maintained. All other factors were also constant except time which naturally varies according to the thickness of the part and the term of pregnancy.

This standard distance of 36 inches does admit of some distortion as compared to a teleo or 2 meter distance. However, with the technique employed this distortion is not sufficiently great to necessitate the consideration of using a larger target distance with its subsequent increase in exposure. If necessary the distortion co-efficient can be calculated and the symphyses can be measured. Therefore, it did not seem advantageous to use special measuring devices such as have been described for work of this character.

The question arose as to the effect of the repeated exposures to the roentgen ray. In no instance was any effect found in either mother or fetus that could be attributed to the use of the roentgen rays. This is in accord with the work of Stein and Arens.

RESULTS

Considered roentgenologically, there are two general types of symphyses pubis. The one is the type found predominatingly in the males. Here the joint is long as the result of the increased height of the pubic bones. The transverse diameter in this type is usually quite short resulting in a long narrow joint (Figs. 1 and 2). The other type is most frequent in females. Here the joint is short in its vertical diameter but wider in its transverse diameter resulting in a shallow but broad joint (Figs. 1 and 2). All gradations between these two general types have been found, and it is noteworthy that many of the masculine type are found in the female and many resembling the female type are found in the male. So frequently is this true that it has been found impossible to differentiate accurately from the contour of the symphysis pubis alone. The characteristic pubic angle of the male is an acute angle while the characteristic pubic arch of the female is an obtuse angle.

The facies of the pubic bones show many variations. Some are smooth and regular while others are saw toothed or serrated. The latter are most frequently found in younger individuals of both sexes and probably represent incomplete ossification (Fig. 3). Many show areas resembling rarefaction and bone absorption and others are hook-shaped or S-shaped.

It has been impossible to find any characteristic changes in the symphysis pubis due to pregnancy. Multiparity apparently has no effect on the joint that can be demonstrated roentgenologically (Fig. 12). Nor has it been possible to demonstrate any changes in the joint during pregnancy or the puerperium. Repeated films taken at varying intervals throughout pregnancy as well as postpartum show no changes in the symphysis pubis (Figs. 7, 8, 9, 10, 11, 12). *In no instance in this series of 80 patients followed through the course of their pregnancy, was any change found.*

Mobility of the symphysis pubis was sought for in each examination. In no instance could it be demonstrated roentgenologically, in any of the three positions. Likewise, widening of the joint due to the influence of pregnancy or labor was not demonstrable. The narrow joint of the young primipara (Fig. 10), remained unchanged throughout pregnancy, and the broad joint of a ix-para likewise remained unchanged (Fig. 10). Apparently the individual characteristics of any given symphysis are ordinarily not sufficiently altered by pregnancy to be demonstrable roentgenologically. Instances undoubtedly occur in which the widening of the joint is obvious but they are uncommon. It would seem that the physiological relaxation of pregnancy is a potential widening in preparation for labor rather than an actual widening. The 98 per cent of Snelling's series of 500 cases showing "increased mobility of not more than 3 millimeters" would seem to fall within the range of normal.

The 5 instances of separation of the symphysis pubis during spontaneous labor reported here for the first time, are as follows:

CASE 1. Mrs. L. R., aged 30 years. She had measles, chicken-pox, and pneumonia in childhood. Two previous pregnancies had ended in short, spontaneous, and uneventual deliveries. The last menstrual period began on July 19, 1922, and she entered the hospital on April 23, 1923. The general physical examination was negative, there being no evidence of rickets or other constitutional diseases. Blood pressure and urine were normal. The interspinous diameter was 26 centimeters; the intercristal, 27.5 centimeters; right oblique, 23 centimeters; left oblique, 22 centimeters; conjugata externa, 18.5 centimeters; and the conjugata interna, 12 plus centimeters. The pregnancy was evidently full term and the fetal head was engaged at the onset of labor. The labor was induced by castor oil, 2 ounces, followed in 1 and 2 hours by quinine sulphate, 10 grains. The first stage lasted 5 hours and the second stage 21 minutes with fulminating labor pains and rapid descent and expulsion of the head. Position was right occiput anterior. The child was a large male 50 centimeters long and weighing 4,700 grams. On the third day the patient complained of severe suprapubic pain which was intensified by any movement of the legs. Examination showed some œdema about the symphysis, marked tenderness, and some mobility of the pubic rami. There was no outward rotation of the legs but passive motion produced severe pain over the symphysis. Median pressure exerted over both trochanters simultaneously produced an intensification of the pain. Pulse and temperature were normal throughout the entire puerperium. Immobilization by 3 inch strips of adhesive tape produced such an intensification of the pubic

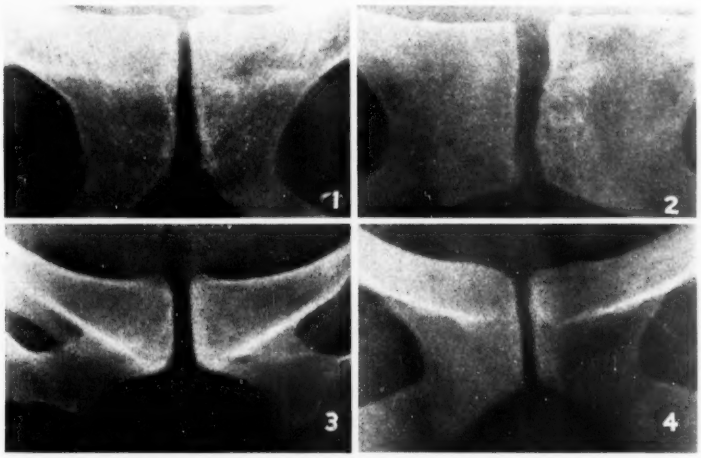

Fig. 1. 1, The deep narrow type of symphysis in the male, type 1, with the typical acute pubic angle. 2, The shallow broad type of symphysis in the male, type 2. The pubic angle here is an obtuse angle forming the pubic arch which is found more frequently in the female. 3, The deep narrow type of symphysis in the female, type 1. Contrast the broad pubic arch with the acute pubic angle in 1. 4, The shallow type of symphysis in the female.

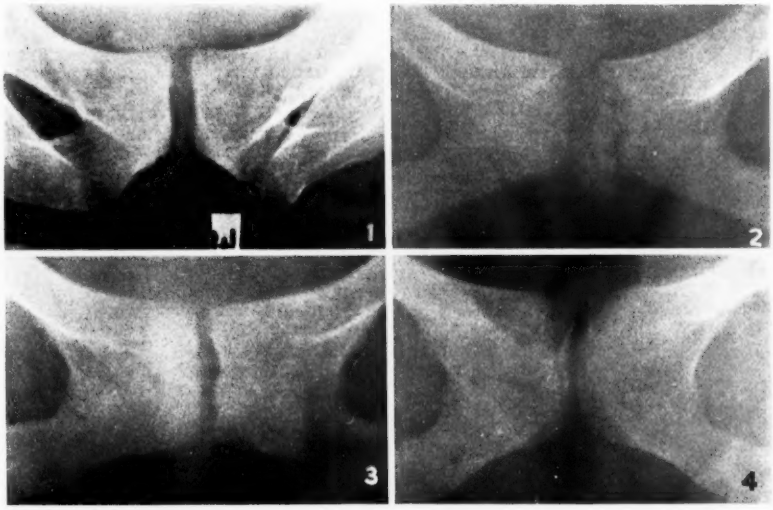

Fig. 2. 1, The shallow, broad type of symphysis, type 2, in the male. Here the pubic bones form a broad arch rather than an acute angle. 2, The deep narrow type of symphysis in the male. Here this male type is associated with a very broad pubic arch. 3, The broad shallow type 2, in the female. Note the marked width of the pubic arch. The angle formed by the pubic bones in this instance is over 135 degrees. 4, The deep narrow type 1, in the female with a typical pubic arch.

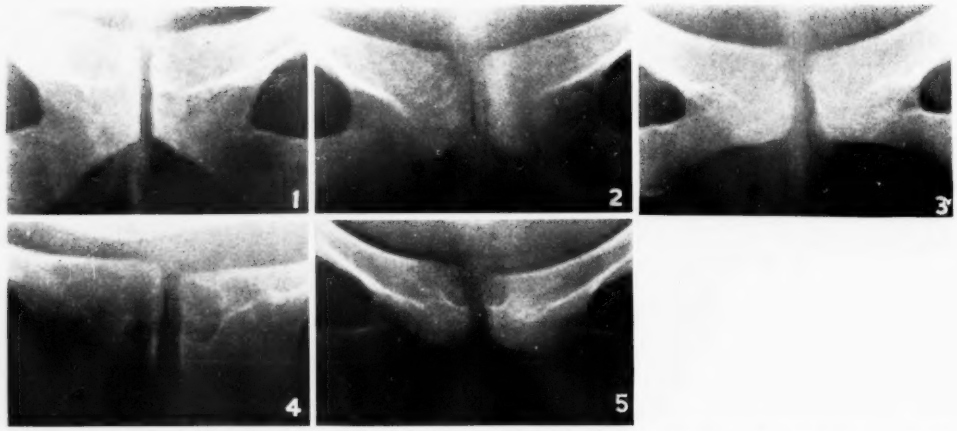

Fig. 3. Variations in the facies of the pubic bones: 1, Smooth type; 2, serrated type; 3, S-shaped type; 4, rarefied type; 5, hook type.

pain that it was necessary to remove them. A vaginal examination at this time showed only a slight gap but definite mobility of the pubic bones. X-ray examination revealed absorption and rarefaction of the left head involving the body of the os pubis but no demonstrable increase in the width of the symphysis. Immobilization was then secured by placing sandbags against the legs from the ankles to the level of the iliac crests. The pain gradually subsided and the patient was comfortable. On the twenty-third day, the sandbags were removed and the patient was able to turn in bed without experiencing any pain. On the twenty-eighth day she was permitted to get out of bed. Walking was painless but the gait was somewhat waddling. This persisted for 2 weeks. The tenderness over the symphysis lasted for 3 months. This patient has since gone through two full term, spontaneous labors which were uneventful and produced no symptoms referable to the symphysis.

Case 2. Mrs. R. J., aged 23 years. There were no previous pregnancies or history of any constitutional diseases. The last menstrual period began March 1, 1924 and labor began on December 9, 1924. General physical examination was negative with no evidence of rickets or other constitutional diseases. The pregnancy was full term, with the head engaged. Pelvic measurements were interspinous, 22; intercristal, 24; right oblique, 21; left oblique, 21; conjugata externa, 21.5; and conjugata interna, 12 centimeters. The first stage of labor lasted for 9 hours and 30 minutes and the second stage for 1 hour and 13 minutes. The delivery was spontaneous after second stage pains of normal intensity and frequency. Presentation was right occiput anterior. The male child was 51 centimeters in length and

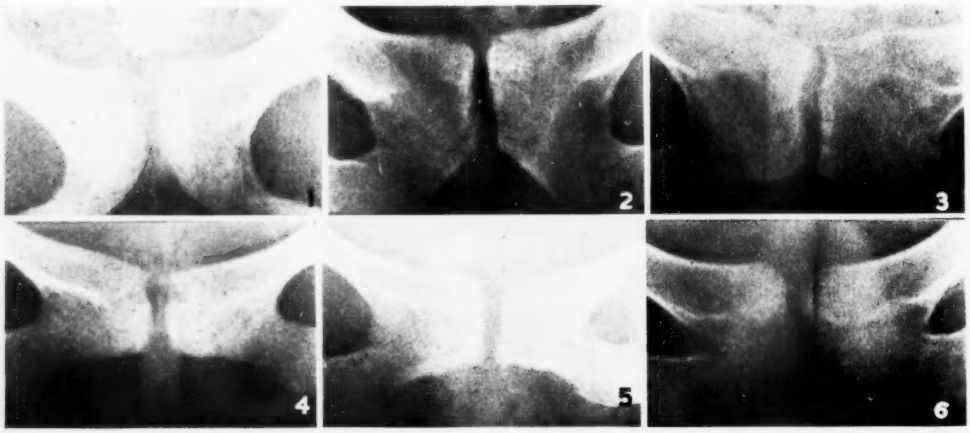

Fig. 4. Dorsal position. Position 1. 1, 2, 3 show three male symphyses taken in the dorsal position. 4, 5, 6 show three female symphyses taken in the dorsal position.

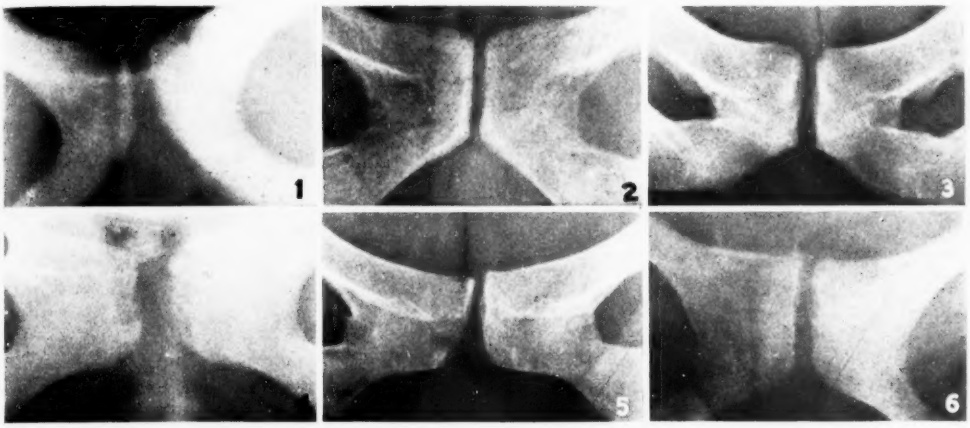

Fig. 5. Ventral position. Position 2. 1, 2, 3 show three male symphyses taken in the ventral position. 4, 5, 6 show three female symphyses taken in the ventral position.

weighed 3,430 grams. The fetal measurements were somewhat large, the occipitofrontal diameter being 12.5 centimeters; the biparietal, 10; occipitomental, 15.5 centimeters. The occipitofrontal circumference was 36 centimeters; the occipitomental, 41 centimeters; and the bisacromial, 34 centimeters.

On the sixth day she complained of suprapubic pain on urination which on the following day was present at all times and was intensified by movements of the legs. Examination at this time showed some œdema of the vulva and symphysis but only slight tenderness. As all symptoms were gone on the eleventh day the patient was permitted to get out of bed. The following day she complained of severe suprapubic pain which prevented her from standing, sitting, or walking, and she was put to bed again. Examination at this time showed tenderness, mobility of the pubic bones which produced a grating sensation, some œdema, and marked tenderness and mobility on vaginal examination. X-ray films showed no increase in width of symphysis. The pelvic girdle was immobilized by circular three-inch strips of adhesive tape which extended completely around the body. This immobilization produced immediate relief. On the twenty-eighth day the patient was permitted to get out of bed. Walking was accompanied by some pain and a waddling gait. Both of these persisted for 3 months. Climbing stairs and any other movements which tilted the pelvis excessively produced pain for about 4 months, after which the recovery was complete. It is of interest to note that the patient had a sturdy but short frame being only 4 feet 9 inches in height. Her husband had an unusually large head and frame, being 6 feet 3 inches in height and weighing 210 pounds.

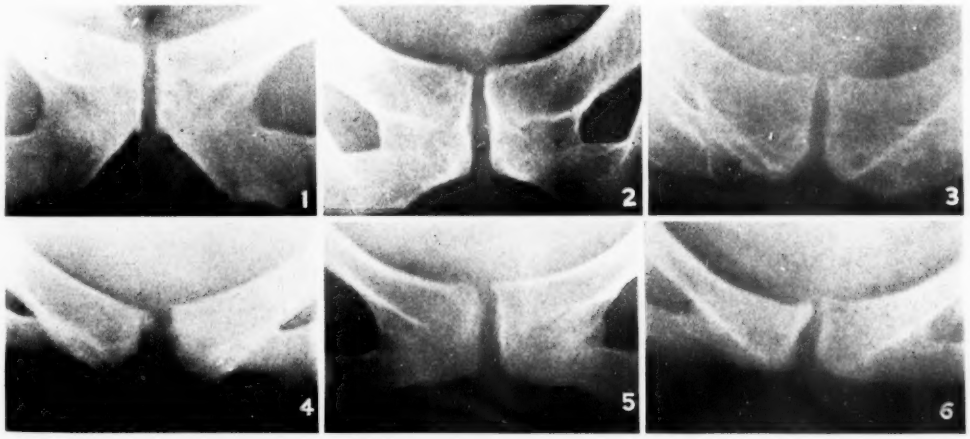

Fig. 6. Semisitting position. Position 3. 1, 2, 3 show three male symphyses taken in the semisitting position. 4, 5, 6 show three female symphyses taken in the semisitting position.

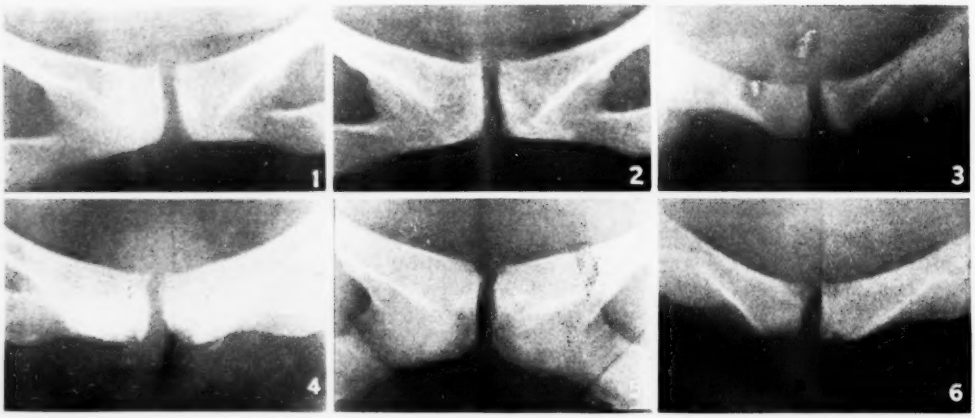

Fig. 7. Symphysis in early pregnancy. The three positions give practically the same contour to the symphysis. 1, 2, 3 are from a iii-para 38 years old. 4, 5, 6 are from a i-para 19 years old.

CASE 3. Mrs. M. W., aged 23 years, with a history of two previous spontaneous and uneventual labors. The last menses began on January 28, 1928, and labor began on November 14. General examination showed no abnormalities or evidences of constitutional disease. Pelvic measurements showed an interspinous diameter of 26 centimeters, an intercristal of 29 centimeters; a conjugata externa of 22 centimeters, and a conjugata interna of 12 plus centimeters. The pregnancy was apparently full term and the presentation was cephalic with the head at the level of the spines when labor began. The first stage of labor lasted 12 hours and 40 minutes, the pains, however, being very slight, ineffectual, and at 10 minute intervals throughout. The membranes were then ruptured artificially and the patient was given 1 minim of pituitrin intramuscularly. The pains then became stronger and more frequent and the baby was born 45 minutes later. The position was right occiput anterior. The child was a male, 50 centimeters in length and weighing 4,170 grams. The puerperium was afebrile but on the eighth day the lochia became foul. On the seventh day the patient complained of pain over the pubis, the right hip, and the lower back. There was a moderate œdema of the labia and the pubic region, marked tenderness over the symphysis and the right sacro-iliac synchondrosis, but no mobility of the pubic bones. A definite gap could be felt which was about 2 centimeters in width. Median pressure exerted over both trochanters at the same time produced relief of the pain. Tilting the

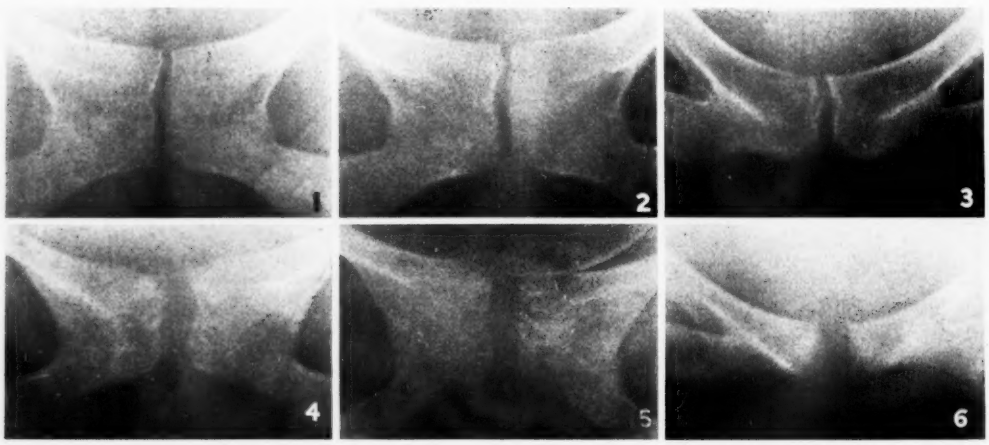

Fig. 8. Symphysis in mid pregnancy. 1, 2, 3 are from a i-para 27 years old. 4, 5, 6 are from a iv-para 34 years old. 6 is markedly narrowed due to the fact that the pelvic inclination in this patient was marked. Being more than normal, the rays caught the pubic bones at exactly right angles to their superior surface. The shadow, therefore, appears quite narrow. In most instances the pelvic inclination was such that some of the superior-inferior length of the pubic bones was projected on the film.

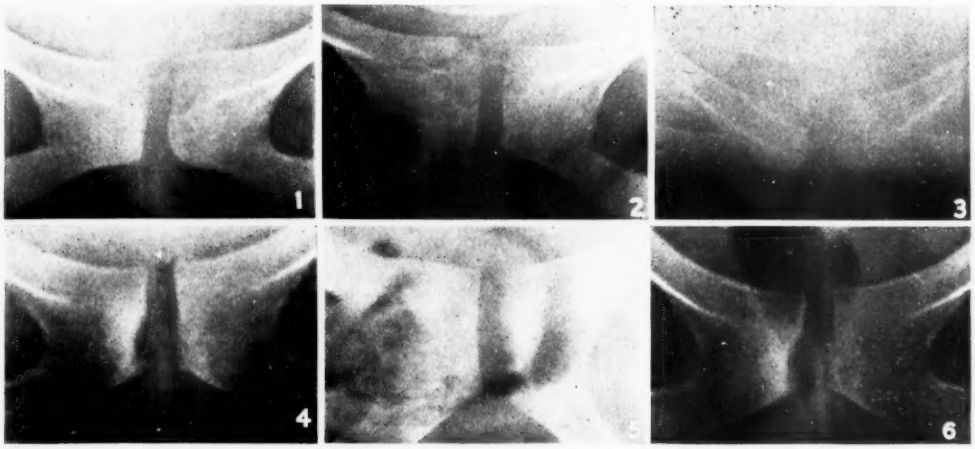

Fig. 9. Symphysis in late pregnancy. 1, 2, 3 show the three positions in a 23 year old ii-para at 8 months. 4, 5, 6 show the three positions in a 27 year old iv-para at 9 months.

pelvis or movements of the legs increased the pain. The pelvic girdle was immobilized by a tight binder which produced relief of symptoms. On the sixteenth day the patient was out of bed but walking was difficult and painful. The gait was unsteady and waddling in character. One week later all symptoms and findings had disappeared and the patient was apparently well.

Case 4. Mrs. B. B., aged 27 years. She had had the usual diseases of childhood and one previous pregnancy which ended by a short labor and a forceps delivery. Last menses began October 4, 1925, and labor began on July 5, 1926. The general physical examination was negative, the blood pressure and urine normal, and the pelvic measurements were interspinous, 27 centimeters; intercristal, 30 centimeters; right oblique, 23.5 centimeters; left oblique, 24 centimeters; conjugata externa, 20.5 centimeters; and conjugata interna, 12 plus centimeters. The first stage of labor lasted 6 hours and 40 minutes and the second stage 35 minutes. The pains were at 3 minute intervals and of average intensity. One hour and 30 minutes before delivery the head was still floating. Delivery was spontaneous and the presentation was left occiput anterior. The child was a male 52 centimeters in length and 3,800 grams in weight. The puerperium was afebrile and uneventual until the eighth day when the patient complained of pain over the symphysis which was aggravated on turning or by moving her legs. Examination showed some tenderness and mobility of the joint but no definite gap could be felt. The mobility was especially marked on vaginal examination which also revealed a 1 centimeter gap. Passive motion of the legs increased the pain. Immobilization was secured by circular adhesive strips which produced relief of symptoms.

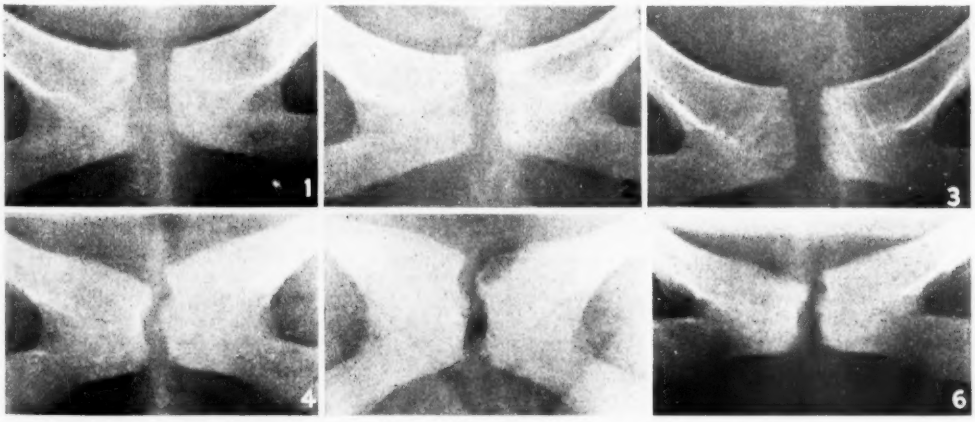

Fig. 10. Symphysis postpartum. The three positions postpartum. 1, 2, 3 in a 17 year old ii-para. 4, 5, 6 in a 21 year old ii-para.

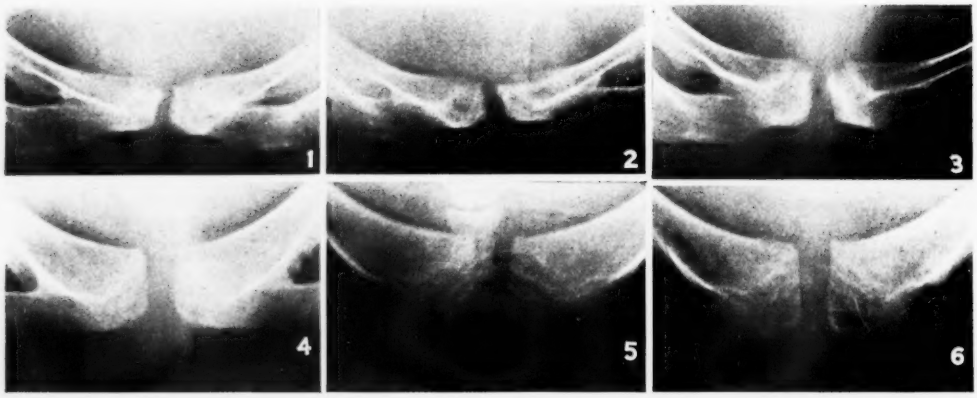

Fig. 11. Symphysis at different times during pregnancy. 1, 2, 3 are from a 23 year old iii-para at 3 and 6 months and postpartum. 4, 5, 6 are from a 16 year old primipara at 3 and 6 months and postpartum. No definite changes can be found during pregnancy or as a result of labor.

On the nineteenth day the patient was allowed out of bed and she walked on the twentieth day. There was only slight soreness over the symphysis on walking. This latter disappeared in 1 week.

Case 5. Mrs. C. A., aged 28 years, had had two uneventual pregnancies and spontaneous labors. Her previous medical history was normal except for the usual diseases of childhood and an appendectomy. Last menstruation began March 1, 1926, and labor began on December 18. There had been slight vaginal bleeding at irregular intervals for the past 5 weeks and the blood pressure on admission was 145/110. General physical examination was negative and the pelvic measurements were interspinous, 24 centimeters; intercristal, 27 centimeters; right oblique, 21 centimeters; left oblique, 21 centimeters; conjugata externa, 20 centimeters; and the conjugata interna, 12 plus centimeters. The first stage of labor lasted 1 hour and 45 minutes with severe pains at 2 minute intervals. The second stage lasted 7 minutes and the delivery was spontaneous, the presentation being left occiput anterior. The child was a female, 50 centimeters in length and 3,005 grams in weight.

On the third day the temperature was 101 F. degrees and the patient complained of severe suprapubic pain. There was marked tenderness over the symphysis, marked œdema of the pubic region and vulva, definite mobility of the pubic bones and passive motion of the lower extremities increased the pubic pain. That night the patient became cyanotic, the respirations became rapid and the following day there was a definite right sided pneumonia. On the fourteenth day a pleurisy with effusion developed. This lasted for 10 days and was followed by complete recovery. The pubic pain had been relieved by immobilization by means of sandbags. X-rays of the symphysis showed no abnormal gap present. The patient was well on the thirtieth day and was discharged. Some tenderness was still present over the pubis and some pain was noticed when she walked. The gait was somewhat unsteady but not waddling in character. Six weeks later the patient was examined and found to be normal.

The 62 case reports found in the literature together with the 5 here reported have been arranged and charted chronologically beginning with Nicholson's report in 1824 (Table I). The most recent report found in the literature is that of Wu in 1930.

SYMPTOMATOLOGY

The time of onset of symptoms referable to separation of the symphysis pubis was recorded in 56 case reports. In 4 patients there were symptoms of pain, discomfort, and difficulty in walking before delivery. Following delivery typical symptoms of separation developed. It would seem that these 4 patients had marked relaxation of the pelvic joints producing symptoms in spite of which traumatic separation, as evidenced by further symptoms and findings, occurred as the result of labor. Eleven women developed symptoms before labor had been completed, 10 immediately following delivery, and 18 in the first 24 hours postpartum. The remaining 13 had no symptoms for the first day or even longer and developed their symptoms as they became more active. Three patients were apparently free from symptoms until they attempted walking or even standing.

The initial symptom of separation of the symphysis pubis is usually the occurrence of pain in the affected joint. Of the 67 case reports, pain is given in 52 as the initial symptom. Seven patients noted in addition to the pain, a cracking or tearing sensation. In 11 other women, the cracking could not only be felt but was recorded as having been audible to the attendants present at the time of delivery.

The predominating symptom of separation of the symphysis pubis is pain. This is located directly over the symphysis and is usually a con-

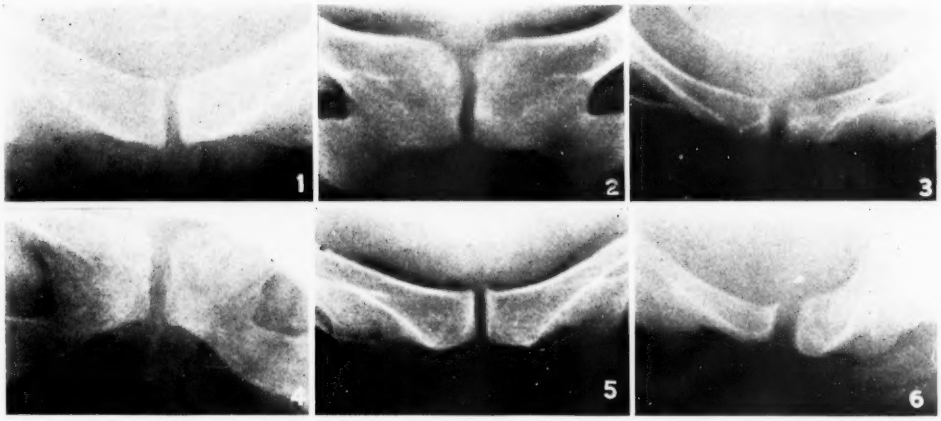

Fig. 12. The symphysis in multiparæ. 1, i-para; 2, ii-para; 3, iii-para; 4, v-para; 5, vii-para; 6, ix-para.

stant dull ache. Any movement of the lower extremities or of the pelvic girdle intensifies this pain until it becomes sharp and lancinating. Turning in bed, use of the bed-pan, sneezing or coughing all increase the severity of the pain. Sitting is usually quite painful and uncomfortable because of the pressure on the lacerated fibrocartilage of the joint. Standing is equally distressing because of the added factor of weight bearing.

Walking produces pain and marked discomfort due to the increased mobility of the joint together with the fact that the two ends of the pubic bones rub against each other. This instability of the pubic arch produces a characteristic gait or "waddle" which persists for some time after the joint has apparently healed. Any movements which tilt the pelvic girdle, such as standing on one leg or the use of stairs, increases the pain in the joint. The typical waddling gait found in this condition is exactly the same as that found after symphysiotomy or pubiotomy. The senior author (J.L.B.) observed and studied the after-effects of these two operations in approximately 50 patients in the Schauta clinic in Vienna in 1907, and he found that the resemblance of the postoperative gait with that produced by traumatic separation is most striking. Equally striking is the similarity of the complications and sequelæ following these two types of lesions.

The severity of the pain together with the fear of intensification on movement results in a pseudoparalysis of the lower extremities which much be differentiated from true paraplegia. Pain is present in the sacro-iliac joints only when these latter are involved. This sacro-iliac pain is never referred to the thighs and does not produce spasm of the hamstring muscles. The tenderness is localized to the joint itself.

PHYSICAL FINDINGS

The physical findings are so typical that "after the first case has been studied, others were diagnosed without roentgenogram" (Wishner and Mayer). Marked swelling and œdema are present about the joint. Tenderness is practically always present and in some instances a definite gap or groove can be felt between the pubic bones. This tenderness is always present on vaginal examination, at which time the gap can be felt, if present. Combined vaginal and external examination often elicits mobility of one or both pubic bones and occasionally such mobility produces a grating sensation. In some instances the pubic bones are no longer in alignment and in addition are found to be at different levels.

In addition to the pseudoparalysis, the lower extremities are usually found in marked eversion and abduction with some external rotation. This latter finding is not, however, as constant as are the eversion and abduction.

All of the typical findings including œdema, swelling, tenderness, disturbed gait, and pain following bilateral pressure on the trochanters were recorded in 28 of the case reports. A gap or groove was felt in 34 but was definitely absent in 12. Movable pubic rami were recorded in 17 and in 5 these were found at different levels. External rotation of the lower extremities was absent in 9. In the 39 reports which did not list all of the typical symptoms, tenderness was recorded in 12, œdema and swelling in 11. In 2 patients the

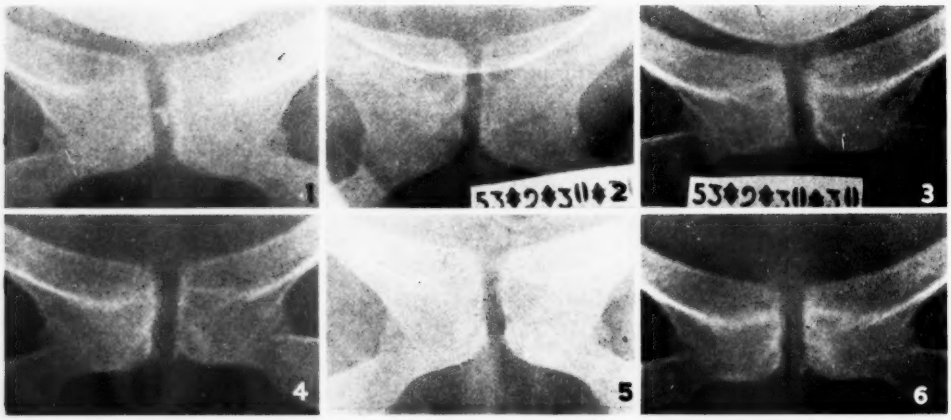

Fig. 13. The symphysis at term and postpartum. The patient is a 27 year old ii-para. 1, 2, 3 are the three positions at term. 4, 5, 6 are the three positions postpartum. The contour of the joint remained unchanged in all three positions.

findings were corroborated on the operating table and in 2 at the postmortem table. Roentgenograms were recorded in 23; in 16 of these the films showed definite separation, and in the 7 remaining no diagnosis could be made from the films.

COMPLICATIONS

Abscess formation following traumatic separation was recorded in 7 instances, 10.4 per cent. Bladder trauma was present in 24 instances, a frequency in 36 per cent. There were 6 recorded deaths in this series, an incidence of 9 per cent. There were 3 deaths due to generalized sepsis following infection and abscess formation in the symphysis pubis, 2 deaths due to miliary tuberculosis following tuberculosis of the traumatized joint, and there was 1 death due to extensive hæmorrhage.

DURATION AND RECOVERY

The duration of this condition was recorded in 45 instances. The symptoms persisted for 1 month or less in 16, 2 to 3 months in 17, 3 to 4 months in 2, 4 to 5 months in 4, 5 to 6 months in 1, and longer than 6 months in 5. Complete recovery was recorded in 35, and a persistence of some symptoms with incomplete recovery in 2. Two other patients were cured by operation.

SUBSEQUENT LABORS

It is of special interest to note the effect of subsequent labors upon symphyses pubis that have healed following rupture in labor. Such a rupture might be presumed to predispose toward rupture in subsequent pregnancies or labors. Apparently this is not the case. In this series, 1 patient had 3 subsequent labors, 2 patients each had 2 subsequent labors, and a fourth patient had 1 labor following the injury to the symphysis pubis. All of these 8 deliveries are recorded as being uneventual and without damage to the healed symphysis.

TREATMENT

The principle in the treatment of separation of the symphysis pubis is the immobilization of the entire pelvic girdle in order to put the joint at rest. In addition the tendency of the pubic rami to pull further apart must be overcome. Sandbags placed against the external surfaces of the lower extremities from the iliac crests down to the ankles will bring about satisfactory immobilization, thus permitting healing and giving relief from pain. The objections to the sandbag method are first that the patient is allowed no movement in bed and secondly that the ends of the pubic bones are not brought together in order to hasten restoration of the torn pubic ligaments.

Broad adhesive strips, overlapping and passing from a line parallel to the posterior superior spine on one side anteriorly around the pelvis to the opposite side, will furnish complete immobilization of the joint in the majority of patients. In addition the patient may move in bed without producing any mobility of the symphysis pubis. Furthermore the lateral pull on the pubic bones is removed, thus permitting more rapid healing of the lacerated ligaments.

A method of applying even more powerful circular compression to the pelvis and to the sym-

physis pubis has been recommended by Wishner and Mayer. The patient is encircled by a broad canvas swathe from behind forward and the two ends are run over pulleys, and weights varying from 5 to 25 pounds are attached. This swathe produces a continuous compressive force in the same arc but in the opposite direction to that of the pathological separation. The authors recommend this form of treatment as it is painless and because recovery is more rapid.

Occasionally healing and recovery of the joint is delayed or even absent. Open surgery of the joint with wiring or suturing of the pubic bones is then necessary. This was done in 2 of the 67 cases collected.

SUMMARY

The world literature to date has yielded only 62 instances of separation of the symphysis pubis during spontaneous labor, to which are added 5 additional case reports from the maternity service of the Michael Reese Hospital.

Of the various hypotheses offered in explanation of the occurrence of this lesion, the most logical is that "rupture of the symphysis pubis is due to marked intensity of the uterine contractions plus marked rapidity of labor." Analysis of the 67 case reports completely supports this theory of etiology, 73 per cent being multiparæ in whom the above mentioned intensity of contractions and rapidity of labor is most apt to occur. Thirty-nine per cent had contracted pelves and 67 per cent had overweight babies. These two factors in the production of disproportion supply additional background for the fulminating type of uterine contractions.

The force that causes the separation of the symphysis pubis is a *wedge effect* produced by the violent thrust of the fetal head through the superior strait. This explanation of separation by a distention force is necessary since the available combined powers of the woman are approximately only one-quarter of the force necessary to rupture the symphysis pubis when applied by a direct pull.

Sacro-iliac involvement occurs when the separation is extensive and involves tearing of the ligaments on the anterior surface of the synchondroses with only tenderness as a possible joint symptom. The right side is most frequently involved.

The bony gap demonstrable by X-ray or by actual palpation is never a criterion of the existence or the degree of the injury nor is it related to the severity or presence of the clinical symptoms and findings.

Separation of the symphysis pubis is rarely complete, a bridge of fibrocartilage nearly always remaining. Hæmorrhage is usual and infection with or without abscess formation occurs when the injury extends into the vagina.

X-ray studies included an analysis of 70 individuals to establish a normal standard and repeated roentgenograms of 80 women at short intervals throughout pregnancy and the puerperium. A technique was evolved, and the semi-sitting posture was found to give the maximum opportunity for studying the symphysis pubis in the three projections.

While there are two generally accepted types of symphysis pubis, the male with deep and narrow joint and the female with the shallow and broad joint, these studies indicate that the sex type cannot be safely diagnosed from the contour of the symphysis pubis alone.

No characteristic changes in the symphysis pubis due to pregnancy could be determined by X-ray. In no instance in this series of 80 patients followed through the course of their pregnancy and puerperium could any definite changes be found in the joint.

Symptoms of separation of the symphysis pubis developed during labor or within the first 24 hours postpartum in the majority of instances. Pain in the affected joint is usually the first as well as the predominating symptom. In addition, a cracking sensation can be felt or even heard in some instances. Any movements which tilt the pelvic girdle increase the pain.

The typical physical findings are diagnostic and include œdema and swelling, tenderness, pain on pressure, and a waddling gait which is characteristic of the condition.

The complications of separation of the symphysis pubis include infection with or without abscess formation, sepsis, hæmorrhage, and failure of union. The mortality rate in the collected cases was 9 per cent. Recovery was complete within 2 months in over 60 per cent.

Separation of the symphysis pubis followed by complete recovery plays no rôle in subsequent labors; neither is the joint affected by subsequent labors.

The proper treatment consists in absolute bed rest with immobilization of the entire pelvic girdle. The simplest type of immobilization is by means of sandbags placed against the external surfaces of the lower extremities and of the hips. Circular adhesive tape may also be used for fixation. An occasional joint requires open operation and fixation. Circular compression by means of pulleys and weights is the most logical form of fixation. It has an especial value in those patients in whom the injury is not recent.

CONCLUSIONS

1. Separation of the symphysis pubis during spontaneous labor is an unusual occurrence.

2. Separation of the symphysis pubis is due to "marked intensity of the uterine contractions and marked rapidity of labor."

3. Multiparity and relative disproportion are additional etiological factors.

4. Separation of the symphysis pubis is a result of the wedge effect produced by the violent thrust of the fetal head through the superior strait.

5. The bony gap demonstrable by X-ray or by actual palpation is never a criterion of the existence or degree of the injury.

6. Sex type cannot be diagnosed from the X-ray contour of the symphysis pubis alone.

7. No characteristic changes in the symphysis pubis due to pregnancy were found.

8. Pain is the initial and the predominating symptom of separation of the symphysis pubis.

9. The characteristic waddling gait is diagnostic.

10. The typical physical findings are œdema and swelling, tenderness, pain on pressure, and the waddling gait.

11. Treatment consists in pelvic immobilization which can be achieved by sandbags, adhesive tape, open operation, or circular compression by pulley and weight.

REFERENCES

1. Ahlfeld, B. Schmidt's Jahrb., 1876, clxix, 185.
2. Idem. Inaugural Dissertations, Leipzig, 1868.
3. Anderodias, J. Bull. Soc. d'obst. et de gynéc. de Par., 1923, xii, 227.
4. Arendt. Quoted by Kehrer.
5. v. Bardeleben. Ztschr. f. Geburtsh. u. Gynaek., 1914, lxxvi, 587.
6. Bauteron. New York M. J., 1878, p. 72.
7. Bazan, J., and Goenaga, J. Semana méd., 1930, i, 88.
8. Benthin, T. Monatschr. f. Geburtsh. u. Gynaek., 1922, lviii, 141.
9. Betz. Quoted by Kehrer.
10. Bochenski. Frommel's Jahresb., 1904, xviii, 1115.
11. Boddaert. Cannstatt's Jahrb., 1853, 342.
12. Brehm, W., and Weirauk, H. V. Am. J. Obst. & Gynec., 1928, xv, 187.
13. Busalla. Zentralbl. f. Gynaek., 1928, lii, 720.
14. Colwell, C. Clinique, 1901, xxii, 653.
15. Cruveilhier. Quoted by Kehrer.
16. Cunningham. Textbook of Anatomy. Philadelphia: Wm. Wood & Co.
17. DeLee, J. B. Am. J. Obst., 1898, xxxviii, 483.
18. Delestre, M. Ann. de gynéc. et d'obst., 1908, v, 228.
19. Denntzer. Arch. f. Gynaek., 1928, cxxxiii, 159.
20. Driver, N. Boston M. & S. J., 1887.
21. Eastman, T. B. New York M. J., 1906, lxxxiii, 130.
22. Eisenberger, F. Zentralbl. f. Gynaek., 1926, l, 2771.
23. Eldridge, E. F. Chicago M. J. & Exam., 1884, xlix, 495.
24. Engstroem. Mitt. a. d. gynaek. Klin. d. O. Engstroem, 1904, vi, 239.
25. v. Fernwald, B. Arch. f. Gynaek., 1894, xlvii, 56.
26. Fessler. Die Festigkeit der Men. Gelenke. Munich, 1894.
27. Fraisse. Quoted by Kehrer.
28. Glenn, J. H. Lancet, 1893, ii, 875.
29. Gmelin, V. Inaugural Dissertations, Tuebingen, 1854.
30. Grenser, D. Monatschr. f. Geburtsh. u. Frauenheilk., 1853, xii, 469.
31. Gusserow. Sitzungsb. d. Gesellsch. f. Geburtsh. u. Gynaek., Berl., 1888, vi, 9.
32. Hartwig, M. Med. News, 1893, lxiii, 220.
33. Heyman, J. Acta obst. et gynec., 1927, vi, 417.
34. Hoffman, G. Neue Ztschr. f. Geburtsh., 1847, xxii, 215.
35. Himmelsbach, G. A. Phila. M. J., 1900, vi, 712.
36. Holzbach, H. Zentralbl. f. Gynaek., 1925, l, 460.
37. Huxley, F. M. J. Obst. & Gynæc. Brit. Emp., 1910, xviii, 327.
38. Jellinghaus. Zentralbl. f. Gynaek., 1899, xxiii, 1305.
39. Kayser, F. Arch. f. Gynaek., 1903, lxx, 78.
40. Kehrer, E. Monatschr. f. Geburtsh. u. Gynaek., 1915, xlii, 321.
41. Keller, R. Gynec. et obst., 1929, xvii, 457.
42. Koestlein. Inaugural Dissertations, Tuebingen, 1880.
43. Kreyberg, K. Zentralbl. f. Gynaek., 1903, 789.
44. Lehman, J. Ztschr. f. Geburtsh. u. Gynaek., 1914, lxxvi, 590.
45. Loeschcke. Arch. f. Gynaek., 1912, lxxxvi, 525.
46. Ludlam, H. Clinique, 1894, xv, 328.
47. Lusena, R. Policlin. (sez. prat.), 1929, xxxvi, 958.
48. Mandruzzato. Ann. di ostet., 1928, l, 1035.
49. Mangiagalli, L. Ann. di ostet., 1887, ix, 196.
50. Mayer, A. Beitr. z. Geburtsh. u. Gynaek., 1906, xi, 200.
51. Meyer, N. E. v. Siebolds J., iii, 10, 36.
52. Montanelli. La Ginecol., 1908, v, 646.
53. Muellerheim. Samml. klin. Vortr., N. N. F. 91.
54. Naujoks, H. Zentralbl. f. Gynaek., 1927, li, 2537.
55. Nemec, E. Bratislavské lekárske listy, 1926, v, 629.
56. Nicholson. Meissner's Forsch. d. Geburtsh., 1824, iv, 72.
57. Olow, J. Acta obst. et gynec., 1927, vi, 423.
58. Pechnikoff, I. Klin. Med., 1928, vi, 1081.
59. Polgar, S. Zentralbl. f. Gynaek., 1925, xlix, 1855.
60. Poullet, J. Lyon méd., 1864.
61. Puech, P. Gaz. d. hôp., Par., 1899, lxxii, 565.
62. Remy, S. Arch. de tocol., 1890, xvii, 698.
63. Retzbach, P. 8, Nuernberg, 1915.
64. Riecke. Heidelberger Klin. Ann., 1831, vii.
65. Rudaux, P. Thesis de doct., 1898, 59.
66. Sauer, K. Wien. klin. Wchnschr., 1899, No. 51, xii.
67. Schatz. Quoted by DeLee.
68. Schauta. Allg. Wien. Ztschr., 1882, 42.
69. Idem. Mueller Handb. d. Geburtsh., 1889, ii, 403.
70. Idem. Wien. klin. Wchnschr., 1899, xii, 904.
71. Schenck, B. Am. Med., 1902, iv, 507.
72. Shordania, J. Zentralbl. f. Gynaek., 1929, liii, 1719.
73. Snelling, F. G. Am. J. Obst., 1870, ii, 561.
74. Stein, I. F., and Arens, R. A. J. Am. M. Ass., 1923, lxxxi, 4.
75. Telfair, J. H. Am. J. Obst. & Gynec., 1923, v, 91.
76. Tuley, H. Am. J. Obst., 1913, lxviii, 852.
77. Walker, J. Am. J. Obst. & Gynec., 1927, xiii, 623.
78. Wishner, J., and Mayer, L. Surg., Gynec. & Obst., 1929, xlix, 380.
79. Wu, L. C. Nat. M. J., China, 1930, xvi, 768.
80. Zulauf. Arch. f. Anat. u. Entwcklngsgesch., von His., 1901, 95.

RECENT DEVELOPMENTS IN THE TREATMENT OF GASTRIC LESIONS[1]

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I SHOULD like to consider this subject from the standpoint of treatment of benign, ulcerating lesions of the stomach and duodenum as well as from that of the treatment of malignant lesions of the stomach of patients of all ages. It might be well, in considering gastric and duodenal ulcers, as they exist in patients in this country, to call attention to a few pathological differences in the two types of lesions. The principle fact in this connection is that ulcerating lesions of the stomach may be ulcerating carcinomata or carcinomatous ulcers, and I know of no scientific way of distinguishing between these two without microscopic examination of the removed lesions. The smallest gastric lesion may still be carcinomatous. I have, on a few occasions, removed carcinomatous lesions of the stomach no larger than 1 centimeter in diameter. Whether these started as carcinomata with areas of ulceration, or whether they started as ulcers and became carcinomatous seems to me to be only of academic interest; the fact remains that the lesions were carcinomatous and were not suspected of being such prior to operation.

ULCER

Ulcers of the first portion of the duodenum, however, practically never become carcinomatous; in fact, so seldom does carcinoma occur in the first portion of duodenum that its incidence there can be regarded as negligible.

It would seem necessary, therefore, to determine accurately the site of an ulcerating gastric or duodenal lesion, for if it is duodenal and produces few symptoms, without complications, a course of medical treatment should be properly carried out before surgical attack is considered. The value of a similar medical regimen for patients with gastric ulcers of small size has been evaluated by Eusterman and Jordan and by others; evidence of healing is disappearance of the niche of the ulcer as seen in the roentgenogram, with relief of symptoms and disappearance of occult blood from the stools. If any one of these three factors persists during the course of a properly regulated medical regimen, carried on over a period of a few weeks, the feeling of both internist and surgeon is that surgery should be done and the ulcer removed.

Surgical removal of such gastric ulcers can be accomplished by cautery excision of the lesion and gastro-enterostomy, or by gastric resection, including the ulcerated lesion in that portion of the stomach removed. Although the recent wave of enthusiasm for gastric resection has prejudiced some in favor of gastric resection for all gastric and duodenal lesions, and although in certain types of lesions, such resections are the procedures of choice, yet it should be emphasized that excision of a gastric ulcer and gastro-enterostomy carries a lower operative risk than gastric resection, and furthermore recurring ulcer following the former procedure is practically absent.

The chronic duodenal ulcer, complicated by bleeding, perforation, or obstruction, is properly dealt with by surgical procedures. On the other hand, a duodenal ulcer without these complications, producing a mild degree of disturbance with little disability, and well controlled by dietary precautions, is a suitable case for a trial of medical treatment. This is particularly true if the duodenal ulcer has been producing symptoms for a short time, usually less than 3 years. In this connection, I think it is worth directing attention to the fact that in the last few years in The Mayo Clinic we have felt that in 55 per cent of the cases of duodenal ulcer a course of medical treatment was worthy of trial, whereas in the other 45 per cent surgical treatment has been considered, without dispute, to be necessary.

Experience has indicated that the proper selection of individual cases will assist materially in the benefits of whatever type of procedure, medical or surgical, is carried out.

Some surgeons, for instance Walton, of London, and Duval, of Paris, are of the opinion that the sooner a patient with chronic duodenal ulcer is operated on, the shorter is the period of disability and the less the necessity of an extensive operation. In further support of this opinion, Duval has expressed the belief that the duodenal ulcer which becomes chronic never heals by any measures other than surgical. Here, again, it would seem that decision as to the proper method of treatment in such cases is an individual problem.

I should like to call attention at this point to the fact that ulcerating lesions of the stomach and duodenum may vary in different races and in different countries. Recently, I have had an opportunity to study gross and microscopic speci-

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mens of ulcerating lesions of the stomach and duodenum found at operation in Germany, Austria, and Hungary. The characteristic features of ulcerations of the stomach and duodenum in these countries are the multiplicity of lesions in both the antrum of the pylorus and in the duodenum; generally, in France and the United States of America, the ulceration tends to localize either to the duodenum or the stomach. In referring to the literature on the subject in German, one is struck by the fact that these lesions are referred to as "peptic ulcers," and such they are, for in practically every specimen associated with one or more duodenal ulcers, there were from three to sixteen superficial yet definite ulcers in the antrum of the stomach. The ulcers of the antrum varied from 0.3 to 1.5 centimeters in diameter, were superficial, and extended to the muscularis mucosa, and associated with them in many instances was extensive gastritis of the antrum. Konjetzny, in his monograph entitled: "The Inflammatory Bases of Typical Ulcer Formation in the Stomach and Duodenum," published colored photographs of gross specimens as well as microphotographs of these lesions. It seems probable, therefore, that pyloroplasty or gastro-enterostomy in these cases is followed by an incidence of recurring ulcer out of proportion to the recurrence in this country, owing to the fact that the stoma of pyloroplasty or gastro-enterostomy would be placed directly in the infected, ulcerating area of the stomach. It is obvious, I believe, that when such diffuse ulceration exists in the stomach and duodenum, resection of the portion containing the ulcerations, by partial gastrectomy, should be the operation of choice.

That most duodenal ulcers occurring in patients in this country are localized to the duodenum, and are not accompanied by ulceration of the antrum of the stomach, can be justified by the observation of most surgeons who have carefully noted the condition of the antrum of the stomach at the time pyloroplasty or gastro-enterostomy has been carried out. Shortly after my return from Europe, I had occasion to resect the stomach of 5 patients with ulcerated lesions. In 2 cases, the resections were done for gastrojejunal ulcers, in 1 of which the complication followed gastro-enterostomy, and in the other, a Devine exclusion type of operation. In 2 other cases, resection was done for a bleeding, perforating duodenal ulcer. In only 1 case of the 5 were superficial ulcers found in the antrum of the stomach in addition to the duodenal ulcers, and in this particular case the ulcers were known to exist prior to operation; diagnosis had been made by the roentgenologist and had been suspected by the clinician. In this case, therefore, with multiple ulcers of the stomach and duodenum, resection of the ulcerated region was indicated. In the 2 cases in which there were hæmorrhagic duodenal ulcers, there was no evidence of ulceration in the antrum of the stomach.

It would seem, therefore, that the problem of duodenal ulcer which concerns the surgeon in this country is whether definite, grossly visible ulceration exists in the antrum of the stomach, associated with duodenal ulcer. If not, conservative procedures, such as excision of ulcers, and pyloroplasty and gastro-enterostomy, are indicated. In the experience of the clinic, such procedures are carried out with a mortality of less than 1 per cent, and with permanent cure in more than 90 per cent of cases. If the ulcerations are diffuse, and if they involve the stomach and duodenum, partial gastrectomy is advisable. Because of the fact that partial gastrectomy for ulcer carries a higher mortality than the conservative operations of pyloroplasty and gastro-enterostomy, the burden of proof rests on the surgeon who advises subtotal gastrectomy for duodenal ulcer.

Why one type of lesion should be present in central Europe and not in Paris, and in but few cases which we have seen at the clinic, is difficult to determine. This difficulty of interpretation is not to be wondered at when it is considered that doubt still exists as to the etiology of duodenal and gastric ulcers, even though a period of 35 years has elapsed since surgical treatment has been recognized as being of utmost value in the handling of most of these cases. Yet, when there is unanimity of opinion regarding the value of a certain operation among certain surgeons in any country for the treatment of a particular disease, it can be said that the results justify such a procedure. In the large clinics in Germany, Vienna, and Budapest, all surgeons of whom I know perform gastric resection for the ulcerating lesions which have been mentioned. In Paris, where evidence seems to indicate that the lesions also are not diffuse, procedures, such as pyloroplasty with removal of the duodenal ulcer, or gastro-enterostomy, are the general rule, except in one large clinic. In the United States, although a few surgeons have advocated gastric resection, as a routine for duodenal ulcers, an overwhelming majority favors selections of the procedure indicated, whether it be gastro-enterostomy, pyloroplasty, or gastric resection.

In evaluating the results following the surgical treatment of duodenal ulcer, criticism has arisen, largely due to two factors: (1) ulcers may recur following operative procedures, and (2) inasmuch

as the etiological factors concerned in the formation of ulcer are not definitely known, it is stated that surgical procedures should be delayed as long as possible. In reply to the criticism concerning recurring ulcer, the statistics would seem to indicate that among patients in this country, who have been subjected to conservative operations for duodenal ulcer, such as pyloroplasty and gastro-enterostomy, the incidence of recurring ulcer is less than 3 per cent. This is true of any group reported or studied which consists of the usual type of American patient, a mixture of various races of the world, in fairly good health, except for the ulcer, living under good hygienic conditions, and eating according to a normal well balanced diet. If a higher incidence of recurring ulcer occurs, it is usually among certain races, and it is possible that in such a group the ulcerations more nearly approach those of the German speaking people.

In considering the uncertainty regarding the etiological factors concerned in the formation of ulcer, it might be well to recall that similar circumstances exist in cases of cholecystitis and renal calculi, and also in fibro-adenomatous hypertrophy of the prostate gland. Compare the etiological factors concerned in these conditions with those of gastric and duodenal ulcer. Does the balance weigh any more favorably in either direction as to results of treatment, or does the incidence of recurrence vary appreciably? I am sure that the general opinion is that the scale is in almost perfect balance, and yet, brilliant results are obtained in the treatment of such lesions when operative procedures are indicated for the removal of lesions or for the relief of obstruction.

I do not think apology is needed for the results obtained in surgical treatment of gastric and duodenal ulcer if indications for operative procedures are present, and if proper operative procedures have been carried out. In the few cases in which gastrojejunal ulcers develop, it often will be found that the original duodenal ulcer, for which gastro-enterostomy had been performed, has healed, and that, after removal of the gastrojejunal ulcer, normal continuity between the stomach and duodenum or intestine can be restored with the expectation that as satisfactory a result will occur, without any greater incidence of recurrence, as after the non-operative treatment of duodenal ulcer. The only difference is, of course, that in the surgical case, when such procedure is carried out, the surgeon knows whether the duodenal ulcer is healed. In other cases, after removal of gastrojejunal ulcer and closure of the openings in the jejunum and stomach, pyloroplasty, with removal of the original ulcer, may be advisable. In some cases of extensive gastrojejunal ulceration, or in cases in which the original ulcer does not appear to have healed properly, or, in healing, has narrowed the first portion of the duodenum so that satisfactory pyloroplasty cannot be carried out, gastric resection is indicated. Such procedure in the treatment of gastrojejunal ulceration at the clinic in 1930 carried a mortality of 3.5 per cent.

The duodenal ulcer which bleeds is worthy of special attention. Such ulcers may be multiple, but if the ulcer is single it is usually situated on the posterior wall of the duodenum and perforation has taken place into the pancreas. A small blood vessel in the crater of such a perforating ulcer usually will be found to be the cause of the hæmorrhage. It has been my custom in such cases, whenever possible, to remove or destroy the lesions, either by cautery or resection of the ulcer. If resection is advisable, pylorectomy, combined with resection of as much of the lower end of the stomach as is thought desirable, reconstructing the anastomosis to resemble that of the Billroth I procedure, has served admirably. When the ulcers have been small, and have been situated only in the anterior wall, excision, combined with one of the plastic procedures, has worked out very satisfactorily. Unfortunately, in many cases of bleeding duodenal ulcer, the large size of the lesion, the subacute inflammation, and the immobility of the first and second portions of the duodenum, make excision or resection of this portion of the duodenum exceedingly hazardous. In these cases, the patient is much better off, and certainly the surgeon is assuming much less risk with gastro-enterostomy, which will prevent recurrence of the bleeding in 80 per cent of these cases, than if a direct attack on the lesion is attempted. Should bleeding recur after gastro-enterostomy, a secondary operation always can be undertaken at which time it is usually possible to remove the bleeding, ulcerating portions much more easily and much more safely to the patient, because of relief of the inflammatory œdema, by proper drainage of the stomach, brought about by the previous gastro-enterostomy.

CARCINOMA OF THE STOMACH

There is no need, I believe, to review the various types of restoration of gastro-intestinal continuity after resection of the stomach. It might be of interest, however, to know that the posterior Pólya, the anterior Pólya-Balfour, and the Billroth I procedures are most commonly used at the clinic.

The Pólya type of operation is widely used in this country, but the Billroth I operation has not

been so extensively employed. There has been hesitancy in carrying out the Billroth I operation because it was originally discarded by Billroth, owing principally to what he thought was a dangerous suture angle formed at the point of meeting of the three lines of suture in the anastomosis, as well as to the fact that little has been written of the indications for the operation or the results following its use in different types of cases. In the last few years I have used this method of gastric resection in more than 50 cases, both for carcinoma of the stomach and for gastric or bleeding duodenal ulcers.

The Billroth I operation consists of resection of the portion of the duodenum containing the lesion, if an ulcer exists, or of that portion of the stomach containing the ulcer or carcinoma, with direct anastomosis between the upper end of the stomach and the duodenum; the stomach is narrowed by suture to the extent that the opening in it is similar to that of the duodenum. The essentials and safety of the operation are dependent on their being sufficient normal stomach above the gastric lesion to be resected so that the anastomosis can be made to the duodenum without tension, and exposure of the first and second portions of the duodenum can be made with sufficient ease to make such anastomosis accurate. In cases in which the caliber of the duodenum has appeared to be small, I have not hesitated to extend the excision down its anterior wall, thus giving as large a size to the anastomotic opening as is desired. Illustrative of results of the operation are 4 cases of carcinoma, graded 1 or 2, without lymphatic involvement, in which the growths were removed successfully and gastroduodenal continuity restored by the Billroth I procedure.

The oldest patient was in her seventy-eighth year, and was operated on in March, 1930; she has been perfectly well since the operation. This patient had gastric retention, owing to the site of the lesion at the pylorus, and quantities of gastric content, varying from 1,200 to 1,500 cubic centimeters, could be recovered. It was necessary, because of the size of the carcinoma, to remove three-fourths of the stomach. This case illustrates the fact that indications for the operation depend not particularly on the amount of stomach removed, but on the necessity of the anastomosis between the end of the stomach and duodenum being made without tension. Whenever preoperative obstruction has occurred in such cases, particularly in elderly patients, I have felt it advisable, in addition, to perform temporary jejunostomy for feeding, which gives complete control of the patient's nutrition. The 3 other patients, all aged more than 70 years, returned home in good condition.

Total gastrectomy. Attention should be called to the fact that total gastrectomy could probably be done in more cases. Such a possibility supports the surgeon in his decision to advise exploration in cases of carcinoma of the stomach regardless of its extent as evidenced by roentgenological examination, provided the condition of the patient permits. Not infrequently a lesion reported as inoperable by the roentgenologist, because of its extent, is found to be removable.

Total gastrectomy has been performed ten times at the clinic; five operations were performed in the last 3 years and three of these in the last year and a half. Mention is made of this because of the fact that the use of spinal anæsthesia and a left rectus incision have permitted more easy exposure of the lower end of the œsophagus than of the fundus of the stomach. In such cases, the question frequently has been raised as to the effect of loss of the entire stomach on the general physiological processes. In this respect, it may be noted that one of the patients successfully operated on by W. J. Mayo lived for almost 4 years. Moynihan and Brigham each reported a case in which the patient was living and well for more than 2 years following the performance of a total gastrectomy.

Physiological and chemical studies following successful total gastrectomy, which I performed September 10, 1929, carried out over a period of 4 months subsequent to operation, did not reveal any appreciable change in the number of erythrocytes, in the concentration of hæmoglobin, in the carbon dioxide combining power, or in the concentration of blood chlorides of urea. A second case in which I operated successfully, December 30, 1930, is being studied similarly, to serve for comparison with the other cases. At the present time (August 24, 1931) the patient is in good condition.[1]

Presence of metastasis. The presence of metastasis in the cervical lymph nodes (so called signal glands), or in the region of the umbilicus when the nodes are definitely proved to contain carcinomatous cells by excision of one of them and its microscopic examination, is in general a contraindication to operation. The same general statement applies to pelvic implants palpated on the rectal shelf on rectal examination. On the other hand, the presence of a small tumor in the liver, existing with a carcinoma of the stomach, should not be taken to be a metastatic nodule unless proved so by microscopic examination of a section

[1] Patient well and without recurrence, June, 1932.

removed from it. Furthermore, I am not convinced that metastasis in the liver of a patient who is in good condition should prevent palliative removal of an ulcerated, infected gastric lesion. There is no reason why such a necrotic, sloughing tumor should not be removed, and actual or impending obstruction relieved, if by such a procedure the remainder of the patient's life may be made more comfortable. I know of several such patients who have lived more than 3 years and been able to carry on their work with comfort. In this connection it should be remembered that death due to metastasis to the liver is usually painless, probably because the metastatic hepatic lesions do not break down or become infected. Whether such palliative resection should be done is a decision to be made in each case; yet the principle established by Sampson Hanley in his treatment of carcinoma of the breast, in my opinion applies equally well to treatment in cases of gastro-intestinal carcinoma, which briefly is that the field should be extended not only to include early cases of malignancy, but also late cases and recurring cases, if by so extending it the remainder of the patient's life can be made more comfortable and can be greatly prolonged.

BACTERIAL CONCRETIONS IN THE KIDNEY PELVIS

With Report of Two Personal Cases[1]

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OCCASIONALLY there are met in the literature reports in which so called bacterial or fibrin concrements are described as found in the kidney pelvis either at autopsy or operation. Such masses are soft and friable and consist of a nucleus or layers of fibrin with perhaps a few crystals among them. Gage and Beal collected a series of soft fibrinous kidney concrements in 1908 and the literature contains some isolated reports since then.

Apart from these fibrinous calculi, however, I wish to report here on another much rarer type of soft kidney stones, namely concrements composed entirely of bacteria. Only a very small number of cases of this type have been reported and these I will refer to later on. In Israel's book, *Chirurgie der Niere und des Harnleiters*, 1925, p. 234, four such cases are mentioned. These concretions are described as "soft, elastic bodies varying in size from that of a bean to a cherry, occurring in the pelvis of the kidney; round, oval or tetrahedraform in shape, and composed entirely of coli bacteria."

I have had two personal cases of this latter nature, the histories of which are as follows:

Case 1. A woman, aged 34 years, had had a feeling of discomfort in the right loin for 12 years. During the last 5 years she had definite attacks of pain in the right kidney region that were occasionally associated with chills and fever. The pains were usually of short duration, of moderate intensity, and were frequently followed by the passage of one or more stones. Altogether she had passed about thirty stones, most of which were about the size of a bean. They were soft and elastic and could easily be crushed between the fingers. Four years ago she had a right nephrotomy, at which time a single large stone was removed (details as to the size and consistency of this stone were not available). She had a very stormy convalescence following her operation which did not interrupt the passage of stones, as she passed several 3 days after the operation and has continued to do so every few months up to the present time. During the past 5 years her urine has been cloudy and contained pus, but she has had very little bladder pain or frequency of urination. For the past 6 months she has lost about 15 pounds, had almost constant pain, and was able to do but very little work.

Physical examination revealed an emaciated woman, weighing 105 pounds. The right kidney, which could be palpated, was somewhat tender. There was a short, puckered scar in the right costovertebral area.

Laboratory data. The urine contained a large amount of pus and blood. The 2 hour intravenously injected phthalein return was 50 per cent. A roentgenogram of the kidneys and bladder revealed numerous indefinite shadows in the region of the right kidney (Fig. 1). The shadows varied in size from ½ to 2 centimeters; they were confined in a small, definite area, similar to the localized clumping of gall stones. Several of the shadows were lamellated but they were without the outside dense ring so commonly seen in gall stones. An X-ray of the biliary tract after the ingestion of gall bladder dyes revealed a normal appearing gall bladder separate from the shadows previously noted.

Cystoscopy revealed an almost completely functionless right kidney. The function of the opposite kidney was normal. A right pyelogram showed an enlarged, distorted pelvis containing numerous areas of lesser density corresponding to the shadows seen in the original X-ray picture (Fig. 2).

Operation. A right posterolateral incision was made below and posterior to the previous incision. The kidney was readily delivered but the convex border was markedly adherent to the pleura. It was necessary to use sharp dissection to free these adhesions; the capsule in one area was stripped from the kidney and a portion left adherent to the pleura.

The kidney was soft, pyonephrotic, and contained only a small amount of functioning tissue. The pelvis was dilated and thickened and contained what seemed to be a soft elastic mass. No definite stones could be felt. The renal vessels and the ureter, which was thickened and dilated, were clamped and cut and the kidney removed.

The patient recovered readily from the operation and had a normal convalescence.

Pathological report. The removed kidney was normal in size and lobulated. The capsule was densely adherent in several places on the convexity of the kidney. When the kidney was opened, the pelvis was found to be considerably dilated, and the mucous membrane was thickened. There were several scars in which the cortical substance was destroyed. The pelvis was almost completely filled with soft, friable, grayish-yellow stones; there were about twelve of these stones measuring from 0.5 to 2 centimeters in diameter. Most of the stones had flattened surfaces due to pressure (Fig. 3). The outer surfaces of the stones were smooth and rubbery and the flattened surfaces almost resembled the faceted surfaces so commonly occurring with gall stones. The stones could readily be crushed with forceps or flattened between the fingers. Examination of the soft elastic material composing these stones by placing a portion in a drop of water and observing microscopically showed large numbers of bacteria (bacillus coli) with a few red blood cells and leucocytes. Culture of the same material showed a growth of bacillus coli with occasional colonies of streptococci. The entire stones were composed of bacteria. Examinations or cultures from either the surface or the center of cut or crushed stones revealed the same condition.

The kidney parenchyma was moderately fibrous. Sections showed large scars in the cortex in which there were many fibrotic glomeruli, atrophy of the tubules, heavy round cell infiltration, and more or less hæmorrhage. Between the scars the glomeruli were congested and the tubular epithelium showed a marked granular swelling. The pelvic mucosa was thickened and heavily infiltrated with round cells (Fig. 4).

[1] Read before the annual meeting of the American Association of Genito-Urinary Surgeons at Buck Hill Falls, Pennsylvania, June 4 to 6, 1931.

Fig. 1. Multiple shadows in right renal area.

Fig. 2. Stone masses showing in pyelogram as areas of decreased density.

The important point in this case to which attention is invited is the nature of the pelvic concretions. The density of the stones was less than that of the pyelographic material. This is rather an unusual occurrence and indicated that only a small amount of shadow-casting material was present. Usually traces of calcium, sodium, and phosphate are found.

A nephrectomy was done as it was impossible to remove all the soft, putty-like material, and this kidney was functionless and not essential. The opposite kidney was entirely sufficient to support life. The patient lived in an inaccessible district where it was difficult to obtain medical care. If the kidney had not been removed and a more conservative procedure attempted, it might later be found necessary to resort again to surgery.

CASE 2. A woman, aged 61 years, had had an occasional attack of pain in the right loin during the last 5 months. The pain usually lasted from 1 to 2 days. Her last attack began 5 days ago. It was of moderate intensity, fairly persistent, and was associated with a generalized jaundice. She was slightly nauseated and had moderate frequency of urination. Previous to the past 5 months she had always considered herself well except that, for the last 20 years, she had had twinges of pain and a sense of discomfort under the right costal margin. She had a good appetite, could eat any food without distress, and did all of her house work.

Examination revealed a thin, small woman with a definite generalized jaundice. The right kidney was tender, easily palpated, and freely movable. The liver edge could be felt below the costal margin and was somewhat tender.

Laboratory data. The urine contained a large amount of pus, some bile, a few red blood cells, and a trace of albumin. The 2 hour intravenous phthalein return was 47 per cent. X-ray examination of the kidneys and bladder revealed a somewhat indistinct shadow 2 by 3 centimeters in the region of the right kidney.

Cystoscopy. The urine from the left kidney was clear but that from the right was cloudy and contained pus and blood. The 15 minute phthalein return was 2 per cent on the right side and 14 per cent of the left side. A right pyelogram included the shadow seen in the original X-ray picture. As the patient was recovering from her pain, she was kept waiting for 2 months before any surgical procedure was attempted, during which time her jaundice cleared up completely, she gained weight, and felt in much better health.

Operation. The kidney was delivered through a right posterolateral incision. It was about 5 centimeters below the normal location and moderately adherent to the overlying fat and to the peritoneum, though quite freely movable. The liver extended down about 3 centimeters below the costal border. When the peritoneum was stripped, there was considerable stippling of the renal surface. The cortex was hard and firmer than normal kidney tissue, suggesting an extensive fibrosis. The kidney was about normal in size but contained very little tissue which appeared or felt normal. The kidney pelvis was slightly dilated, thickened, and the ureter was 1 centimeter in diameter, with thickened, stiffened walls. The pelvis was opened and a large amount of soft, putty-like stone material extruded. There was also a large amount of mucus and necrotic tissue expelled through the opening. The vascular pedicle and ureter were clamped, cut, and tied separately and the kidney was removed.

Fig. 3. Soft, faceted stones in kidney pelvis. On section some show a lamellated structure.

Pathological report. The specimen consisted of a small kidney measuring 9.5 by 4.5 by 3 centimeters. The pelvis was moderately enlarged and contained five soft, light brown stones. These were very soft so that they could be crushed between the fingers. Examination of the material composing these stones, by placing some in a drop of water and examining microscopically, showed large numbers of bacteria (bacillus coli). Cultures taken from the surface of the stones or from the central portions of crushed stones revealed a growth of bacillus coli. The mucous membrane of the pelvis was distinctly thickened and congested. The renal parenchyma was pale, firm, and rubbery and apparently markedly fibrotic.

Sections showed a very diffuse, fibrous, extensive atrophy of the tubules; many glomeruli were surrounded by fibrous tissue. There was an extremely heavy round cell infiltration throughout. At one place in the pelvis was a circumscribed fibrous area that appeared to be an organized thrombosis. In places the tubules were hypertrophied.

In this, as in the previous case, it was possible to obtain active colon bacilli or a positive culture from any part of the stones either on the surface or from the cut section. In both cases the kidneys were partially or almost completely destroyed and in such a condition that it would be impossible to obtain a permanent cure by attempting to remove the stone fragments.

Due to the lack of normal kidney tissue or function on this side, the displacement of the kidney, the extensive fibrosis, the patient's age, and because of the normal function of the opposite kidney, a nephrectomy was done.

The definite jaundice that was seen 2 months before operation was probably due to the downward pull of the heavy fibrotic kidney on the biliary passages. It has been shown that a right movable kidney drawn forward and downward causes traction on the duodenum at a point 2 or 3 centimeters below the entrance of the biliary tract; and, though it does not occlude the lumen, it may interfere with the contents of the bowel sufficiently to impair digestion and to cause biliary obstruction. Some investigators believe that the jaundice associated with movable kidney comes from a pull on the fixed, descending part of the duodenum and obstruction of the papilla of Vater.

REVIEW OF LITERATURE

The cases mentioned in Israel's book are those of Schmorl, Jores and Neumann. In Schmorl's first case the autopsy of a 58 year old woman, who died of lung disease but showed evidence of cystopyelitis, disclosed a bacterial (bacillus coli) calculus in the kidney pelvis. In Schmorl's second case a woman of 81 years gave a history of cystitis. Following a kidney colic, bean-sized bacterial (bacillus coli) calculi were passed 4 days before death. Jores found the same picture *intra vitam* in urine and in a kidney colic. In Neumann's case pea to cherry-size concrements were found embedded in pus in the pelvis of a woman of 26 years who had had a nephrotomy. Microscopically and culturally these concrements were found to be composed of bacillus coli. Later, this patient showed a right sided pyelonephritis.

Bornemann described a case of a girl aged 7 years with a history of whooping cough and colic. At the age of 4 years she had hæmaturia and intense pain. Blood clots were passed at the end of urination. The urine showed much albumin, pus, bacteria (cultured as bacillus coli) and casts, and X-ray examination disclosed shadows in each kidney pelvis. The child was treated medically and died. At autopsy a hard stone was found in each kidney pelvis, besides a large number of soft, laminated, spheroid concrements, the largest, walnut-sized. Microscopic examination showed that the soft laminated concretions in both pelves were composed entirely of bacillus coli, the segmental layers containing leucocytes, red blood cells, and necrotic epithelial cells.

In a case reported by Ward, the patient was a woman of 35 years who had had malaria and typhoid as well as a history of bowel disorder. Fifteen months before coming to Ward she had had a severe right renal colic with hæmaturia which lasted a month, then another colic on the same side, accompanied by acute renal infection. X-ray examination showed a calculus. About a month later, that is about 3 months after the

onset of symptoms, there were several mild attacks of colic during which forty-one "soft" stones were passed. Subsequently, the calculus seen by X-ray was eliminated, together with three others of the "soft" variety. Nearly 100 more soft stones were passed during the following 10 months. Ward found that a pyelogram showed the right kidney dropped and ureter kinked below the pelvis. Some months later there was another attack of persistent pyuria and obstruction of the right kidney with signs of inflammation, and the kidney was removed. The urine had shown abundant bacillus coli. The patient recovered. The removed kidney contained over one hundred calculi which, on examination, were found to be of laminated structure and composed almost entirely of bacteria more degenerated toward the internal part of each lamella. From the chemical examination the nucleus of these calculi seemed to be a minute blood clot with the outer layers composed entirely of bacteria. The cementing layers appeared to be of a mucoid nature which, Ward presumed, was the factor necessary for cohesion.

Kelly and Dible observed a case in a woman of 42 years. Her symptoms dated back to the age of 8 years, when she was treated for some urinary condition. A few years later she had a definite attack of pyelitis which involved the right kidney and, about 10 years or so later, an attack of painless hæmaturia.

X-ray examination disclosed nothing, but by the cystoscope the presence of a calculus in the right ureter was diagnosed. Occasional attacks of hæmaturia followed but later X-ray examinations failed to locate the stone again, and it was presumed to have disintegrated. Bacillus coli and blood were found in the urine which also contained casts composed chiefly of bacteria. Exploration of the right kidney pelvis resulted in the removal of masses of calcareous and soft, friable material from the kidney pelvis (calcium oxalate and fibrin). This woman was ultimately nephrectomized in 1929 (15 years after the last cleaning out of the kidney pelvis), on account of agonizing pains and loss of right kidney function. The whole of the kidney pelvis and calyces were tightly packed with soft, faceted, dark olive green masses. In some cases there was a very minute calcareous nucleus. Microscopically the concretions were composed of bacillus coli and a reticulum substance of the nature of fibrin which was about the masses of bacteria. The authors, however, think that this was not fibrin but largely composed of degenerate leucocyte cells. There was some kind of stratification of the outer layers, but this was entirely lacking toward the center of the concretions.

Fig. 4. Partially destroyed kidney after removal of stones. Scar of previous nephrotomy indicates destruction of large area of renal parenchyma.

ETIOLOGY OF BACTERIAL RENAL CONCRETIONS

From a consideration of the cases quoted and of the literature, two questions arise in the etiology of this particular type of concrement: first, that of some kind of nucleus; and second, that of some kind of cementing process which causes the masses of bacteria to adhere. There seems to be little doubt that the concretions develop by continual proliferation of live bacteria and the addition of cells to exterior of growing mass.

In regard to the nucleus, Lauda postulated the presence of necrotic shreds cast off from the pelvic wall in pyelitis. Many of the cases gave the history of an old pyelitis, but, if this is to be considered as causative, the occurrence of bacterial concretions should be much more common than they apparently are.

Schmorl thought the first event to be a simple adhesion of bacteria to form small masses. The urine from an infected renal pelvis not infrequently contains clumped masses of bacteria; at times these are sufficiently large to obstruct the ureteral catheter. It seems quite probable that a number of these clumps might coalesce and form the nucleus of a larger mass.

Bornemann discusses the similarity of fibrin stones and bacterial stones and concludes that they are identical. The nucleus of the concrements in Ward's case appears to have been a minute blood clot. Kelly and Dible conclude that the concretions were originally formed about tiny particles of stone or gravel.

The most important factor in these cases, however, appears to be the continuous deposit of bacteria in the kidney pelvis. This point, as well as the fact that the particular bacillus is almost always the bacillus coli, seems to have received very scant consideration from the authors. Ward thinks that in his case the bowel disorder may have been the source of the urinary tract infection and that the process was intensified by the presence of kidney ptosis and ureteral kink. The fact that all the cited cases were in women and that kidney ptosis is more common in the female sex may have some bearing in the matter.

There seems but little to support the theory that the cementing substance in the concretions is of a fibrinous nature; it seems more plausible to consider it to be a degenerated leucocyte product.

In my own two cases there was no evidence of a calcareous or fibrinous nucleus, nor did the urine contain fibrin shreds.

I do not wish to include in this report cases in which bacilli other than the bacillus coli were cultured from the kidney pelvis. Liebermeister mentions 3 cases of tuberculosis of the kidney with calculus in which the tubercle bacillus was found in the stone, and Ferrier and Bliss report a case of pyelithotomy complicated by gas bacillus (bacillus welchii) infection originating in the nucleus of a renal calculus.

CONCLUSIONS

Two personally observed cases of kidney pelvic concretions composed of bacteria (bacillus coli) are reported, and some other cases reported in the literature are discussed. Continuous infection of the kidney seems necessary, but it is likely also that some special circumstance is called for in order that a calculus of this type should be produced.

Bacterial concrements in the kidney pelvis may be concomitant with the usual type of indurated kidney calculus.

REFERENCES

BORNEMANN, W. Die sogenannten "Bakteriensteine" im Nierenbecken. Frankfurt. Ztschr. f. Path., 1913, xiv, 458.

FERRIER, P. A., and BLISS, W. P. Pyelithotomy complicated by gas bacillus infection originating in a renal calculus. J. Urol., 1928, xx, 471.

GAGE, H., and BEAL, H. W. Fibrinous calculi in the kidney. Ann. Surg., 1908, xlviii, 378.

JORES. Cited by Neumann.

KELLY, R. E., and DIBLE, J. H. Massive bacterial concretions in the renal pelvis. Brit. J. Urol., 1930, ii, 143.

LAUDA. Bakteriensteine im Nierenbecken und ihre Entstehung. Frankfurt. Ztschr. f. Path., 1922, xxvii, 263.

LIEBERMEISTER, G. Deutsche Arch. f. klin. Med., 1922, cxl, 195.

NEUMANN, A. Ueber Bakteriensteine im Nierenbecken. Deutsche med. Wchnschr., 1911, No. 32, 1475.

SCHMORL. Verhandl. d. deutsch. path. Gesellsch., 1901. (Cited by Neumann.)

WARD, R. O. Urinary calculi composed of bacteria. Brit. J. Surg., 1926–1927, xiv, 230.

ACUTE HEAD INJURY

A Study of One Thousand Cases[1]

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THIS study of 1,000 cases of acute head injury admitted to Bellevue Hospital represents a group clinically diagnosed "Fracture of the Skull." Head trauma should be classified clinically by its resultant pathology; accordingly the following plan is offered.

I. Simple scalp wounds
II. Skull fracture with or without associated brain injury
  a. vault
  b. base
  c. simple (linear or comminuted)
  d. depressed
  e. compound
III. Brain injury with or without associated skull injury
  a. Concussion
  b. Cerebral œdema
  c. Cerebral contusion
  d. Cerebral laceration
  e. Hæmorrhage-intradural, extradural, or both
IV. Combinations of the above

The material reviewed was admitted for study only on the presence of one or more of the following positive criteria following severe injury to the head: (1) postmortem evidence of skull fracture or brain laceration; (2) positive X-ray evidence of skull fracture; (3) bloody spinal fluid obtained by lumbar puncture.

Mortality rate. The mortality rate of the entire group was 37.8 per cent. This was found to vary slightly from year to year as shown in Table I.

TABLE I.—MORTALITY RATE

	Lived	Died
Mortality percentage.		
1000 cases	62.2	37.8
Mortality percentage according to years.		
1926	62.6	37.4
1927	65.7	34.3
1928	59.4	40.6
1929	60.9	39.1

There appears to be a definite increase in the death rate associated with increasing age. In children under 12 years of age the death rate was 25.8 per cent, whereas in people over 60 years the mortality was 49.6 per cent (Table II).

TABLE II.—MORTALITY RATE ACCORDING TO AGES

Cases	Age Groups	Mortality rate per cent
97	0 to 12 years	25.8
782	13 to 60 years	37.5
121	Above 60	49.6

Types of trauma. Automobile accidents and falls are the commonest causes of acute head injury as shown in Table III.

TABLE III.—TYPES OF TRAUMA

	Cases
Auto	266
Street car	17
Bicycle	2
High fall	86
Low fall	135
Fall down stairs	95
Assault	33
Horse kick	1
Blunt instrument	22
Falling object	21
Flying object	2
Moving object	12
Crushing object	5
"Fight"	18
Unknown	285
Total	1000

Local scalp condition. The associated local scalp condition was reported in 713 of the cases and these findings are grouped as in Table IV.

TABLE IV.—LOCAL SCALP CONDITION

	Right	Left	Both sides	Location not specified	Total
Laceration	149	193	20	107	469
Hæmatoma	47	58	7	42	154
Ecchymosis or contusion	25	31	2	26	84
Œdema	0	3	0	2	5
Abscess of the scalp	0	0	0	1	1
Total	221	285	29	178	713

X-ray evidence of fractured skull. Roentgenograms were made in only 499 of the 1,000 cases. Of these 230 were negative; 186 showed fracture of the vault; 63 showed fracture of the base, and 20 gave evidence of depressed fracture of the cranium (Table V).

[1] Neurological Department (Cornell), Bellevue Hospital, New York City.

TABLE V.—X-RAY EVIDENCE OF FRACTURED SKULL

	Lived %	Died %	Total %	Total number cases
Positive roentgenogram of the vault	17.3	1.3	18.6	186
Positive roentgenogram of the base	5.6	.7	6.3	63
Positive roentgenogram for depressed fracture	1.9	.1	2.0	20
Negative roentgenogram	21.9	1.1	23.0	230
Roentgenogram not made	15.5	34.6	50.1	501
				1000

TABLE VII.—POSTMORTEM FINDINGS

	Right	Left	Mid-line	Side not specified	Total
Fracture base and vault					
Posterior location					
Linear	13	9	2	0	24
Composite	1	7	0	0	8
Lateral location					
Linear	21	23	0	1	45
Composite	13	15	0	0	28
Anterior location					
Linear	2	3	0	0	5
Composite	3	5	0	0	8
Vault fracture					
Linear	16	13	0	3	32
Composite	2	5	0	1	8
Depressed	4	2	0	3	9
Total	75	82	2	8	167

Summary

Fractures described in autopsy	167
Fracture present but not described	82
Autopsy but no skull fracture found	8
Total number of cases with autopsy	257
Percentage of cases with autopsy (378 patients died of the total 1,000)	68.0 per cent
Percentage of autopsies showing a fracture of the skull	96.9 per cent

TABLE VII.—POSTMORTEM FINDINGS (Continued)

Brain

	Right	Left	Both sides	Side not specified	Total
Laceration					
Contrecoup	40	39	1	27	107
Direct	28	31	4	20	83
Undetermined type	0	0	0	46	46
Total	68	70	5	93	236
Percentage of cases with autopsy showing laceration of the brain					91.8%
Hæmorrhage					
Subdural	27	26	27	0	80
Epidural	16	14	5	0	35
Subarachnoid	6	8	10	0	24
Middle meningeal	6	2	0	0	8
Ventricular	1	1	3	0	5
Total	56	51	45	0	152
Percentage of cases with autopsy showing hæmorrhage of the brain					59.1%
Infection (abscess) (1 secondary to frontal sinusitis)	2	0	0	0	2

Meninges

12 leptomeningitis (non-suppurative)—including 1 arachnoid adhesion to brain.
14 suppurative meningitis—2 associated with septic sinuses.

Hæmatoma or brain contusion	3	3	0	5*	11
Herniation	1	2	0	0	3
Compression	1	0	0	3	4

Ventricles—2 internal hydrocephalus (1 with associated ependymitis).
*Including 1 cerebellar.

TABLE VIII.—SITES OF HÆMORRHAGE

	Cases %	Lived %	Died %
Bleeding from at least one orifice (i.e., ears, nose, mouth, or about eye)	70.1	42.5	27.6
No bleeding from orifices	25.1	18.9	6.2
Undetermined (incomplete charts)	4.8	.8	4.0
Bleeding from the nose*	35.7	20.0	15.7
Bleeding from the ear*	34.2	21.1	13.1
Bleeding from the mouth*	18.8	8.7	10.1
Ecchymosis about the eye*	14.9	8.9	6.0
Subconjunctival hæmorrhage*	8.7	5.8	2.9

*Also occurring in combinations.

TABLE VI.—TIME UNCONSCIOUS—MORTALITY—LUMBAR PUNCTURES

	Not unconscious	No mention	Time undetermined	"Few minutes"	"Short"	"Long"	Minutes 1	5	10	15	20	30	60	120
Bloody tap	35L 10D 45T	216L 58D 274T	155L 45D 200T	8L 2D 10T (2*)	16L 1D 17T	36L 173D 209T (84*)	2L 0D 2T	4L 0D 4T	5L 1D 6T	4L 0D 4T	2L 1D 3T (1*)	8L 0D 8T	4L 1D 5T	4L 0D 4T
Clear tap	4L 0D 4T	7L 1D 8T	12L 1D 13T	1L 1D 2T	6L 0D 6T	2L 8D 10T (2*)	0L 0D 0T	0L 0D 0T	0L 0D 0T	1L 0D 1T	0L 0D 0T	1L 0D 1T	0L 0D 0T	0L 0D 0T
Cloudy tap	0L 0D 0T	2L 1D 3T	1L 2D 3T	0L 0D 0T	0L 1D 1T	1L 2D 3T	0L 0D 0T	0L 0D 0T	0L 0D 0T	0L 0D 0T	0L 0D 0T	0L 0D 0T	0L 0D 0T	0L 0D 0T
No tap	21L 1D 22T	26L 21D 47T	16L 4D 20T (1*)	1L 3D 4T (3*)	2L 0D 2T	9L 37D 46T (23*)	0L 0D 0T	0L 1D 1T	0L 0O 0T	2L 0D 2T	3L 0D 3T	5L 1D 6T	0L 1D 1T	0L 0D 0T

Summary

	Lived	Died	Total
Bloody tap	499	292	791
Clear tap	34	11	45
Cloudy tap	4	6	10
No tap	85	69	154
Total number of cases			1000

(Probably small amounts of blood—no organisms reported)

L, Lived; D, died; T, total; *, died without regaining consciousness.

TABLE IX.—TIME IN HOSPITAL

Time in hospital	Lived	Died	Complication	Oper. on, lived	Oper. on, died	Transferred	Left against advice
On admission	..	10	..	..	..	..	..
½ hour	..	25	..	..	..	..	1
½ to 1 hour	..	9	..	..	..	..	..
2 hours	..	17	..	..	..	..	..
3 hours	..	13	..	..	..	..	..
4 hours	..	14	..	..	..	..	..
5 hours	..	8	..	..	..	..	..
6 hours	..	7	..	..	1	..	..
7 hours	..	6	..	..	..	..	..
8 hours	..	6	..	..	..	..	..
9 hours	..	5	..	..	..	..	..
10 hours	..	1	..	..	..	..	..
11 hours	..	3	..	..	..	..	..
12 hours	..	2	..	..	..	..	..
13 hours	..	1	..	..	..	..	..
14 hours	..	2	..	..	1	..	..
15 hours	..	2	..	..	..	..	..
16 hours	..	..	..	..	..	..	..
17 hours	..	3	..	..	..	..	..
18 hours	..	3	..	..	1	..	..
19 hours	..	1	..	..	..	..	..
20 hours	..	1	..	..	..	..	..
21 hours	..	2	..	..	..	..	..
22 hours	..	..	..	..	..	..	..
23 hours	..	..	..	..	..	..	..
24 hours (approximate)	..	38	..	..	5	..	3
2 days	..	57	..	..	4	1	10
3 days	1	28	..	..	4	4	18
4 days	5	20	..	..	4	5	12
5 days	4	17	..	..	..	7	11
6 days	3	9	..	..	..	5	15
7 days	3	7	..	..	1	6	11
8 days	5	5	..	..	..	6	8
9 days	9	5	..	..	1	2	11
10 days	19	3	..	..	1	..	4
11 days	13	7	..	..	..	2	3
12 days	20	4	..	..	..	1	6
13 days	14	4	..	..	..	2	1
14 days	11	3	..	..	..	3	7
15 days	20	..	..	..	..	3	6
16 days	17	..	..	..	..	..	5
17 days	16	..	..	..	..	..	5
18 days	9	1	..	..	..	1	3
19 days	18	1	..	..	..	1	2
20 days	19	..	..	..	..	1	..
21 days	61	2	..	2	..	2	2
22 days	28	..	3	1	..	2	..
23 days	17	..	..	..	..	..	2
24 days	9	1	4	..	..	..	..
25 days	5	..	6	..	..	2	..
26 days	5	1	4	1	..	..	..
27 days	4	..	2	..	..	1	..
28 days	2	..	..	2	..	..	..
29 days	2	..	2	..	..	..	1
30 days	3	..	1	..	..	..	..
31 days	2	..	5	1	..	..	..
32 days	1	..	1	1	..	..	..
33 days	2	..	2	..	..	..	..
34 days	..	..	..	..	..	..	..
35 days	1	..	2	..	..	1	..
36 days	1	..	1	..	..	..	..
37 days	..	..	4	..	..	..	..
38 days	..	..	2	..	..	1	..
39 days	1	..	1	..	..	..	..
40 days	..	..	..	..	..	..	..
41 days	..	..	..	..	..	..	..
42 days	1	..	..	..	..	..	..
43 days	..	..	..	1	..	..	..
44 days	..	1	..	1	..	..	..
45 days	1	..	..	..	..	..	..
46 days	..	..	1	..	..	..	..
47 days	..	..	..	..	..	..	..
48 days	..	..	..	..	..	..	..
49 days	..	..	..	..	..	..	..
50 days	..	..	..	1	..	..	..
51 days	..	..	1	..	..	..	..
52 days	..	..	..	1	..	..	..
53 days	..	..	..	..	..	..	..
54 days	..	..	..	..	..	..	..
55 days	..	..	..	1	..	..	..
56 days	1	..	2	..	..	1	..
57 days	..	..	1	..	..	..	..
58 days	..	..	1	..	..	..	..
59 days	..	..	..	..	..	..	..
60 days	..	..	2	1	..	..	..
Total	353	355	48	14	23	60	147

TABLE X.—COMPLICATIONS

	Lived	Died	Total
Appendicular			
Fracture radius	4	1	5
Fracture ulna	4	1	5
Fracture humerus	5	6	11
Fracture femur	1	7	8
Fracture tibia	8	8	16
Fracture fibula	3	5	8
Fracture patella	3	1	4
Fracture maleolus	2	2	2
Fracture ankle	0	1	1
Fracture hand	1	0	1
Fracture foot	3	0	3
Axial			
Fracture clavicle	23	8	31
Fracture scapula	3	0	3
Fracture vertebra	1	5	6
Fracture pelvis	0	3	3
Fracture ribs	4	25	29
Fracture acromion	1	0	1
Fracture sternum	0	3	3
Face			
Fracture nasal bones	9	9	18
Fracture through orbit	6	11	17
Fracture hard palate	0	2	2
Fracture mandible	2	3	5
Fracture zygoma	2	2	4
Fracture maxilla	3	0	3
Fracture molar	1	1	2
Fracture orbital plate with laceration of 2d nerve	2	0	2
Alcoholism	184	127	311
Shock	60	51	111
Brain abscess	0	3	3
Bullet wound	0	2	2
Pulmonary œdema	1	28	29
Pneumonia	2	70	72
Aspiration of blood into lungs	0	2	2
Hæmothorax	0	2	2
Bronchitis	2	0	2
Fibrinous pleurisy	0	2	2
Emphysema	1	2	3
Fibroid phthisis	1	0	1
Acute suppurative meningitis	0	14	14
Cellulitis of scalp or face	2	0	2
Cellulitis regions other than head	1	0	1
Erysipelas of scalp or face	4	2	6
Erysipelas regions other than head	0	1	1
Lacerated wound of head infected	6	1	7
Otitis media	3	1	4
Ruptured ear drum	6	5	11
Mastoiditis	2	0	2
Blindness due to trauma	2	0	2
Keratitis	1	0	1
Conjunctivitis	3	1	4
Abscess parotid region	1	0	1
Abscess cervical lymph nodes	2	0	2
Other infected foci	2	2	4
Contagious impetigo	1	0	1
Synovitis	1	0	1
Septicæmia	0	1	1
Internal injuries	0	6	6
Ruptured diaphragm	0	1	1
Multiple injuries not otherwise described	2	6	8
Mental symptoms, acute and persisting	8	1	9
Lateral sinus thrombosis (and jugular resection) after mastoidectomy	1	0	1
Abscess of scalp	1	0	1
Gangrene of leg	0	2	2
Conditions existing previous to present illness:			
Epilepsy	10	4	14
Heart disease	6	4	10
Lues	9	1	10
Tuberculosis	0	2	2
Arteriosclerosis	5	8	13
Hypertension	3	2	5
Hernia	3	0	3
Pregnancy	0	1	1
Paralysis agitans	1	0	1
Kidney condition	0	4	4
Liver condition	0	1	1
Arthritis	2	0	2
Encephalitis	0	1	1
Anterior poliomyelitis	1	0	1
Diabetes mellitus	0	1	1
Paralysis of arm with atrophy (following previous injury)	1	0	1

All patients with head injury should have X-ray films of the skull made in at least two positions (i.e., anteroposterior and lateral views).

Spinal fluid findings. In 846 cases (of the 1,000 studied) lumbar punctures were done and reports were made on the spinal fluid. These have been tabulated in relation with reported time periods of unconsciousness and mortality (Table VI). From this it is clear that the longer the period of unconsciousness, the poorer is the prognosis as regards life. Of 791 cases in which the spinal fluid was bloody, 292 lived and 499 died. Many patients died before lumbar puncture was performed (154).

Postmortem findings. The postmortem findings by the New York City Medical Examiners have been tabulated according to the plan outlined by Dr. B. M. Vance.[1] Of the 378 patients dying because of head injury, autopsy examinations were made in 257. Of these, 96.9 per cent showed fracture of the skull and 91.8 per cent showed laceration of the brain.

Bleeding from orifices. Bleeding from the ears, nose, mouth, or about the eyes occurred in 701 of the cases. An analysis of the sites of hæmorrhage is given here.

Time in the hospital. The attached grouping of the patients' time in Bellevue Hospital, correlated with the ultimate result and the presence of complications, is most interesting. Of the 378 patients who died, 201 passed away within the first 24 hours in the hospital—this argues for early medical therapy. In this series all patients operated upon within the first 10 days following injury died. (Cases with obvious subdural hæmatoma, operated upon, are not included in this series.)

Complications. It is sufficient here to give a listing of the recorded complicating conditions. Alcoholism is very frequent, having been present in 311 cases. Pneumonia occurred in 72 cases, and 70 died. Meningitis (acute suppurative) occurred in 14 cases; all succumbed. Brain abscess occurred in only 3 cases and was fatal.

TABLE XI.—NEURAL FINDINGS

Mental States

	Cases
Alcoholism	176
Coma	194
Confusion	35
Psychoses (disoriented, irrational, etc.)	104
Hyperirritability	117
Stupor	173
Normal	175
Dead on admission	25
Not recorded	1
Total	1000

[1] Arch. Surg., 1927 xiv, 1023.

TABLE XI.—NEURAL FINDINGS (Continued)

Cranial Nerves

I Nerve

Only 3 cases with recorded involvement.

II Nerve

	Bilateral	Right	Left	Total
Congestion of the disc	75	3	2	80
Papillœdema	20	2	1	23
Hæmorrhage	1	4	2	7
Normal	..	..	..	129
Not observed (i.e., not recorded)	..	..	..	736
Left hospital or died before observation	..	..	..	25
Total	96	9	5	1000

II. Nerve involvement of the 239 cases observed:

Congestion of the disc	33.5 per cent
Papillœdema	9.6 per cent
Hæmorrhage	2.9 per cent
Normal	54.0 per cent

III, IV, VI Nerves

Size

Dilated	102
Contracted	85
Right greater than left	121
Left greater than right	99
Unequal	17
Normal	410
Not observed	149

Reaction

Sluggish	135
Fixed	154
Normal	422
Not observed	289

Extraocular movements

Diplopia	5
Restricted upward gaze	2
Left internal strabismus	7
Left external strabismus	4
Right internal strabismus	6
Right external strabismus	7
Bilateral internal strabismus	5
Bilateral external strabismus	3
Conjugate movement to left	8
Conjugate movement to right	14
Conjugate movement upward	1
Left strabismus (?)	2
Strabismus	10
Skew deviation	1
Nystagmus—spontaneous lateral	13
Nystagmus—spontaneous to right	5
Nystagmus—spontaneous to left	2
Vertical nystagmus	0
Nystagmus undetermined plane	43
Rotary nystagmus	1
Ptosis—right	6
Ptosis—left	13
Exopthalmos or endopthalmos	0
Total ophthalmoplegia	1
Left eye, no movement	1
Corneal ulcer	1

V Nerve

Motor

Right	0
Left	0

Sensory

Right	2
Left	6

VII Nerve

Peripheral nerve paralysis (discreet)

Right	51
Left	50

Supranuclear facial weakness associated with hemiplegia on same side

Right	14
Left	14

TABLE XI.—NEURAL FINDINGS (Continued)

VIII Nerve					
Deafness					
Right					15
Left					18
Bilateral					1
Weber, referred to					
Right					4
Left					6
Rinné					
Right					4
Left					4
Bilateral					1
Tinnitus					
Right					2
Left					1
Bilateral					6
IX Nerve					
No recorded involvement.					
X Nerve					
Inability to swallow					1
XI Nerve					
No recorded involvement.					
XII Nerve					
Tongue deviation to					
Right					7
Left					7

Motor Power

Motor palsies—W, weakness, paresis; P, paralysis; S, spasticity; F, flaccidity

Monoplegias					
Arm	Right	W	6		
		P	1	14	
		S	5		
		F	2		27
	Left	W	3		
		P	3	13	
		S	6		
		F	1		36
Leg	Right	W	1		
		P	0	4	
		S	3		
		F	0		9
	Left	W	2		
		P	0	5	
		S	3		
		F	0		
Hemiplegias					
Right arm and leg		W	11		
		P	5	24	
		S	5		
		F	3		41
Left arm and leg		W	6		
		P	3	17	
		S	6		
		F	2		
Paraplegias (both legs)		W	0		
		P	1		12
		S	8		
		F	3		
Quadriplegias		W	4		
		P	2		
		S	35	Probably mostly alternating phases of decerebration	
		F	36		
Decerebrate fits (alternating flaccid and spastic states)					5

Summary	
Total number of cases with motor palsy	171
Negative (no abnormal motor signs)	315
No observations of motor status recorded	514
Total	1000

Of 486 cases with observations of motor status,
35.2 per cent showed motor palsy,
64.8 per cent were negative.

Co-ordination

Gait impaired	
Falling to left (residuum)	1
Finger to nose and heel to knee—no observations made.	
Speech	
Motor aphasia	13
Sensory aphasia	2
Stuttering (?)	1
(No observations made in most cases.)	

Reflexes

Deep reflexes	
Changed	588
Normal	222
Not recorded	190
Number of cases recorded	810
Percentage of recorded cases with changes in deep reflexes	72.6 per cent
Superficial reflexes	
Changed	435
Normal	211
Unrecorded	354
Number of cases recorded	646
Percentage of recorded cases with changes in superficial reflexes	67.3 per cent

Sensory

29 cases of the 1,000 fractured skulls showed sensory changes.

Abnormal Involuntary Movements

Opisthotonus	
Convulsions	
General	81
Focal	34
Athetosis	1
Tremor	
General	2
Focal	23
Twitches	
General	2
Focal	4
Total	148

Neural findings. Only 181 of the 1,000 cases had no evidence of involvement of the nervous system. Complete neurological examinations were not possible (and were not made) in all patients; however, the compilation (shown in Table XI) was possible.

TABLE XII.—TREATMENT

	Lived	Died	Total
Pressure reducing			
Repeated spinal tap	95	70	165
$MgSo_4$	509	150	659
Caffeine	80	86	166
Caffeine, aspirin, phenacetin āā grs. V solution	70	1	71
Glucose (hypertonic)	19	45	64
Sedative			
Paraldehyde	109	59	168
Magendie's solution (morphine)	206	131	337
Luminal	184	27	211
Veronal	38	4	42
Trional	7	1	8
Allonal	3	0	3
Codeine	68	22	90
Bromide grs. X, chloral grs. X	139	26	165
Chloral hydrate	10	6	16
Bromide	18	8	26
Hyoscine	24	24	48
Phenacetin	14	0	14
Chloroform	1	2	3

TABLE XII.—TREATMENT (Continued)

Cardiac			
Digitalis	12	29	41
Glonoin	1	0	1
Strophanthin	0	5	5
Gastro-intestinal			
Rhubarb and soda	16	0	16
Deod. tri opii	4	1	5
Lockwood's gastric sedative	1	0	1
Mineral oil	11	0	11
Olei ricini	6	0	6
Croton oil	1	0	1
Respiratory			
Oxygen	0	1	1
Artificial respiration	0	4	4
Alpha lobelin	0	1	1
Anti-luetic			
Mixed treatment	4	0	4
Miscellaneous			
Atropine	23	71	94
Adrenalin	11	89	100
Pituitrin	0	3	3
Saline clysis	22	48	70
Glucose-nutritive	11	37	48
Gastric lavage	8	20	28
Spiritus frumenti	30	22	52
Acetyl salicylate	22	0	22
Strychnine	8	3	11
Dakin's	1	0	1
Insulin	3	4	7
Glycerin	5	0	5
Tonic, iron, quinine, strychnine	3	0	3
Nutritive rectal tap	1	1	2
Potassium iodide	5	1	6
Imperial drink	1	0	1
Sherman's vaccine	0	1	1
Cerium oxalate	1	0	1
Blood transfusion	1	3	4
Phlebotomy	0	1	1
Ammonium chloride	2	0	2
Fibrogen	3	4	7

Treatment and operations. Treatment in this group was found to be most varied. The listing includes therapy directed both toward the acute phase following injury and for underlying or associated existent complications. It should here be pointed out that this material represents hospital admissions during the years 1926 to 1929. Since that time the routine "skull case" treatment has included administration of hypertonic glucose by vein, to all patients.

Surgical operations on the skull were performed in 37 cases (including one simple trephine); of these 14 lived and 23 died.

TABLE XIII.—OPERATIONS

Type		Cases
Trephine		1
Sub-temporal decompression		
Right	18	
Left	14	
Both sides	1	
Side not given	3	
Total		36*
Osteoplastic flap		0
Sub-occipital decompression		0
Total number of operations		37

*Of these 8 had a depressed fracture.

CONCLUSIONS

This study reveals the frequency of central nervous system involvement complicating acute head injury. It is statistically proved that conservative management of this group of cases is indicated.

A simple plan of treatment has been described in part elsewhere[1] and consists of the following steps:

1. Treatment of shock by the intravenous injection of 100 cubic centimeters of 50 per cent hypertonic glucose solution.

2. Lumbar puncture for diagnosis and treatment.

3. Repetition of hypertonic dextrose by vein to reduce increased intracranial pressure (100 cubic centimeters of 50 per cent solution, 3 times daily).

4. Injection of caffeine sodiobenzoate, 7½ grains (0.5 grams) every 4 hours (hypodermically).

5. Rectal taps of 25 per cent solution of dextrose, 4 ounces (120 cubic centimeters) every 4 hours.

6. Elevation of head of bed 15 to 45 degrees.

7. The carrying out of operative procedures indicated in compound fractures which require débridement, and in cases suspected of progressive middle meningeal hæmorrhage.

8. The use of antimeningococcus serum in suitable cases.

9. The performance of right subtemporal decompressions in comatose patients with marked papillœdema, who do not respond to the aforementioned procedures within 3 hours.

10. Uncomplicated depressed skull fractures may be elevated after the acute stage of shock has passed. Surgical interference in this group may often be safely postponed for many days.

We wish to express our thanks to the directors of the Four Surgical Divisions at Bellevue Hospital for their courtesy in permitting us the use of this material; and to Miss Sarah P. Shiras for her untiring industry with these records.

[1] Foster Kennedy and S. Bernard Wortis. J. Am. M. Ass., 1931 xcvi, 1284.

SUNLIGHT IN SURGERY

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THE value of strong natural sunlight in the prevention of infection and treatment of wounds was first called to attention while practicing in the Atacama desert which makes up the greater part of the Province of Antofagasta in Northern Chile.

This is a region of very intense sunlight and practically no rain—drier even than the better known Sahara desert. The amount of sunshine in this part of the Chilean Pampa from the Pacific Ocean to the west ranges of the Andes Mountains is an almost constant quantity. In all seasons it can be depended upon with almost absolute certainty. Expensive equipment was therefore not necessary in order to take advantage of its therapeutic benefits. It became our custom to expose badly mangled hands, arms, and legs to the sterilizing influence of the ultraviolet rays contained in this hot, white sunlight. The effects were so unmistakable as to leave no room for doubt. The devitalized tissues of the wounded members became mummified. Débridement operation could be done later under the guidance of an absolute line of demarcation. No other method of antiseptic treatment, in my experience, has been anything like as effective or as satisfactory as this simple and inexpensive use of natural sunlight. However, its use is not general even in this region and little has been noted of its beneficial effect.

A few years later while visiting in Quito, the capital of the Republic of Ecuador, we learned something more about the use of sunlight from Dr. Isidro Ayora, later the distinguished president of that country, and his notable colleague, Dr. Villavicencio Ponce. These able surgeons had a keen appreciation of the value of natural sunlight. They had arranged circular plantings of shrubs in the grounds surrounding their clinic. There was an opening through which the bed of the patient could be passed. The interior of this green circle formed a cubicle in which the patient could lie naked with the whole body exposed to the healing rays of the equatorial sun. They explained that they had been using the sun rays very much as the writer and his predecessor, Dr. William F. Shaw, now of Mexico City, had been using them in the Chilean desert. They considered it the most effective treatment for the superficial lesions of leprosy, a disease unfortunately only too common in that region. They observed that it was even more effective than the vaunted chaulmoogra oil treatment.

Although the vogue of sun bathing has begun to spread, it is remarkable how little the sun rays have been used in this country and it is still difficult to get a real open-air sun bath even in the summer time, the only season when the ultraviolet content would be strong enough to be effective in the northern latitudes. In places where the sun is a more dependable agent such as Miami Beach, Florida, a real interest in this subject has begun to develop and some valuable studies are now being made by O. J. Seitlein at the St. Francis Hospital and at the laboratory established at the University of Miami through the vision and generosity of Dr. Thompson.

In the North, especially in the large cities, it is still a difficult matter to obtain a location and privacy where real sunlight exposure of the whole body is available.

While not minimizing excellent results and benefits to be obtained by intelligent use of the various sources of ultraviolet rays that are available, it has always been a source of regret that the beneficial rays of natural sunlight are not more extensively used.

The more powerful therapeutic lamps may be a source of considerable mischief in untrained hands; their use must be intelligently supervised. Recently there has come to our attention, a lamp which approximates natural sunlight so closely that it may really be considered an effective substitute. It has the further merits of being almost if not entirely free from danger in general use by nurses or orderlies in the wards of the hospitals. The lamp devised by Dr. Matt. Luckiesh and his associates, in the research laboratory of one of the great electrical associations, consists of a heavy tungsten filament in a bulb of special glass with a button of quicksilver which volatilizes. The volatilized mercury then shortarcs the tungsten filament producing a mercury arc which emits practically the spectrum of the mid-day summer sun, including its ultraviolet content.

The technical description, which shows how by screening, changing voltage, etc., the emanation may be moved up and down on the spectral ladder to whatever level is required, is not properly a part of this paper. The diagram better illustrates the spectral level of optimal benefit (Fig. 1).

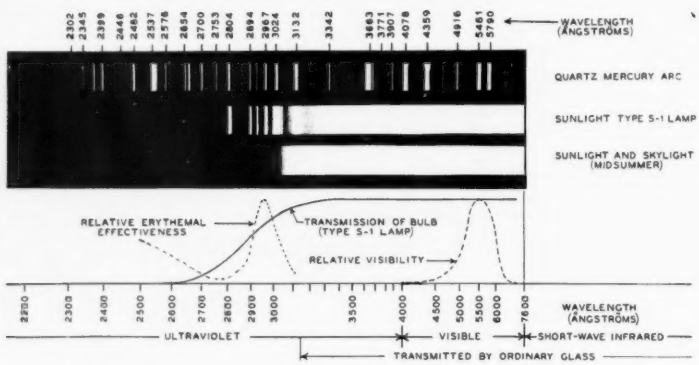

Fig. 1. Comparison of spectra of the quartz mercury arc, sunlight lamp, and midsummer sunlight. The spectral regions of erythemal effectiveness, ultraviolet radiation, visible radiation, and transmission of ordinary glass are shown.

During the past year we have had four of these lamps installed in the wards of St. Alexis Hospital and have made some clinical observations as to the uses to which this artificial sun might be put in the surgical field.

The devices are light and easily portable and we have had no trouble or difficulty in moving them around from one part of the hospital to another, so that certain types of cases might be treated in which we wish to observe the effect of their use (Fig. 2).

We had been using the quartz light in certain selected cases, especially in plastic work as a prophylactic against infection and welcomed the opportunity to try this simpler and more foolproof device, especially as we felt that it more nearly approached the natural sunlight with which I had experience in South America.

There is a special difficulty in keeping certain types of surgical wounds free from infection. There are difficulties encountered in keeping certain skin areas sterile. This may be on account of their position, as for example, the perineal region in the female. In the repair of a relaxed vaginal outlet, the area is moist, contamination is ever present, and resulting infection of the postoperative wound following perineorrhaphy, for example, threatens the success of all plastic procedures in this region. In this type of case the "sun lamp" has been very beneficial. An application two or three times a day, starting within 24 hours after operation, has kept the wound dry and inhibited the growth of bacteria. These wounds have done well under the treatment; better than formerly. Whether this has been due only to a negative action in inhibiting growth of bacteria or whether there has also been a stimulating effect such as increased blood supply thus aiding repair, is yet to be seen. At any rate, it seems to have had its greatest value in such cases. The lamp has also been used on other surgical wounds, such as laparotomy wounds, slow in healing, small plastic skin wounds, accidental lacerations, skin grafts. In all there have been about two hundred cases that have been partially treated with the lamp. By that is meant that all routine measures were used in addition to the lamp.

As a means of controlling infection, it has many advantages: it is clean; it does not stain; it is not greasy or sloppy, and it is usually painless.

The method of using the lamp has been very simple. We have started exposure of the wound area with the bulb at a distance of about 30 inches for 5 minutes, as shown in the illustration (Fig. 3). This is repeated in 8 hours. Each succeeding day the time has been increased 1 or 2 minutes, depending upon the degree of pigmentation present in the skin. The distance is also gradually decreased but is never made less than 20 inches. No attempt has been made to protect the adjacent areas. They are exposed along with the area that is being treated. The ordinary reaction has been a very mild erythema on the first day, with no subjective symptoms of burning or itching, followed by a light tanning after three or four days' exposure. We have had only two unfavorable results from the use of the lamp. The first was a Reverdin pinch-graft on the thigh of a boy aged 7 years. These grafts had taken and were growing well when, hoping to speed their growth, the lamp was used in the usual manner with the result that the grafts sloughed off, and it was necessary to apply other grafts. In another case—a very painful sprained shoulder—the lamp was used and produced a sedative effect, relieving the pain but producing a severe sunburn. This was not a surg-

Fig. 2. Illustration shows the lamp in use in infected compound fracture.

Fig. 3. Sun lamp—initial treatment given 24 hours after perineorrhaphy.

cal case of the type to which we have been referring but we relate it because of the lesson it teaches, namely, that occasionally a patient's skin is extremely sensitive to the ultraviolet rays.

The results of the study of the grades of pigmentation in the normal skin, which is being made by Dr. Thompson, at Miami Beach, will be very useful in enabling us to avoid such unpleasant experiences.

The lamp had its most spectacular and beneficial effect in a case of tuberculous peritonitis. Very often after operation in such cases the wound fails to heal or a sinus forms. In our case the lamp was used immediately and prompt healing of the wound followed; no sinus formed and apparently the lamp had a beneficial effect upon the peritonitis. In this case the lamp was continued for a period of 4 weeks. When its use was discontinued the abdominal wall was tanned to a dark, almost chocolate, brown color, but the patient had never experienced any discomfort.

This paper, based on limited clinical observation, is issued as a preliminary report with the hope of stimulating further observation and the use of this therapeutic agency in purely surgical conditions. We are absolutely convinced that it has definite value as a prophylactic and bactericidal agent, and that we have by the invention of this lamp obtained a simple and inexpensive method of approximating the effects of natural sunlight. It is almost but not quite foolproof. The same thing can also be said with regard to natural sunlight itself. However, any intelligent nurse or orderly can be taught the safe use of this lamp after a very brief instruction. The lamps are portable and can be kept on the ward or in various parts of the hospital and applied very much as other routine procedures of nursing care.

We shall be interested in reading the experiences of other surgeons as we feel that there is a wide possibility for the use of this agency in our special field.

HÆMANGIOMA OF THE VERTEBRÆ

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HÆMANGIOMA of the vertebræ is very uncommon. We have had occasion recently to see a case in which it was possible to make a pre-operative diagnosis from roentgenograms. The patient was successfully operated upon and was very much improved following operation.

HÆMANGIOMA OF EIGHTH THORACIC VERTEBRA WITH SIGNS OF CORD COMPRESSION AND EVIDENCE OF COMPLETE SUBARACHNOID BLOCK. TYPICAL ROENTGENOGRAPHIC APPEARANCE. OPERATION FOLLOWED BY MARKED IMPROVEMENT.

The patient, a Jewish woman, aged 46 years, was admitted to the service of Dr. W. G. Spiller in the Hospital of the University of Pennsylvania on December 12, 1930. She came in complaining of inability to walk, a difficulty which was traced back to 1926, when following the death of her husband she became weak in her legs, and encountered difficulty in moving about as easily as formerly. Her weakness became gradually worse until about 5 weeks before admission, when she found she was unable to walk without assistance. For 2 weeks before admission she was unable to walk at all.

When examined on admission, the only positive findings were present in her nervous system. There was a paralysis of the lower extremities, with signs of a level lesion extending to the ninth thoracic segment. The legs were utterly powerless and paralyzed. The right thigh seemed atrophic anteriorly, especially in the region of the abductors. The patellar and Achilles reflexes were increased. There was a Babinski on the right side but none on the left. Pain sensation was lost over both lower extremities to about the twelfth thoracic. Heat and cold were lost in the same area. Vibration was lost to the eighth thoracic vertebra.

Spinal puncture was done. There was an initial pressure of 4 millimeters of mercury, a level which did not change with compression of the jugular veins. There was evidence therefore of a complete subarachnoid block, although the vague sensory level would hardly have led one to expect it. There were 11 lymphocytes in the spinal fluid and the Wassermann and colloidal gold were negative.

A roentgenological examination of the entire spine made December 17, 1930, showed a lesion involving the eighth thoracic vertebra. At first the appearance was interpreted as an irregular mottling involving the body and lamina. The sagittal view (Fig. 1) showed in addition a very confusing soft tissue shadow mainly to the left of this vertebra which suggested either a neoplasm or cold abscess resulting from tuberculous disease. The final identity of this shadow was never determined. Further study of the appearance of this vertebra especially in the lateral view (Fig. 2) suggested that the appearance might be due to enlarged vessel channels in the bone, and one of us, having seen the original roentgenograms of Bailey and Bucy's case, suggested the possibility of the lesion being a hæmangioma of the vertebra. A subsequent examination after campiodol injection between the third and fourth lumbar vertebræ showed, with the patient in the inverted position, a complete block to the opaque fluid at the intervertebral space between the eighth and ninth vertebræ. It is quite significant that the operative notes on this case stated that "there was not a suggestion of anything which would account for even a partial block, much less a complete one." This finding possibly may be explained by the fact that there had been a lessening of symptoms between the times of examination and operation.

Because there was no clear cut sensory level, a campiodol injection was made January 21, 1931, in view of the suspected spinal cord tumor. The iodized oil stopped definitely at the level of the intervertebral disk between the eighth and ninth thoracic vertebræ (Fig. 3).

The patient developed a retention of urine, but before operation she voided of her own accord and her legs became more mobile. There was no change in her sensory findings. A spinal puncture repeated at this stage demonstrated no subarachnoid block, but there were 36 lymphocytes in the spinal fluid. This partial remission of pressure manifestations was regarded by Spiller as characteristic of hæmangiomata of the spine. It may also account for the fact that while the campiodol roentgenographic study showed a definite block at the time, there was no evidence of block found at the time of operation, over 3 weeks later.

Operation. A decompressive laminectomy was performed by Dr. C. H. Frazier, February 13, 1931, with the removal of the spinous processes of seventh, eighth, ninth, and tenth thoracic vertebræ. At operation it was noticed on rongeuring the laminæ that the bone was very much more vascular, softer, and more cancellous than normal. This was especially true of the eighth and ninth thoracic vertebræ. Upon removal of the laminæ, there was noticed between the laminæ and the dura a layer of highly vascularized tissue which appeared very much like a granuloma. Subdural exploration was entirely negative, and no evidence of block could be found. The patient made an excellent recovery following the operation, regained much power in her legs, lost her sensory disturbances, and was able to walk out of the hospital. Three months after her discharge she was seen in the follow-up clinic and was found to be entirely recovered. Her gait was perfectly normal, and she showed no evidence of either motor or sensory paralysis. Roentgentherapy was instituted later in an attempt to prevent any further encroachment by the growth.

The microscopic picture showed a typical hæmangioma of the capillary and cavernous type (Fig. 4).

The tissue which was removed from the spinous process and lamina of the vertebræ was composed of capillary and cavernous spaces lined with endothelium and often filled with blood. No myelogenous tissue was present. Among the blood spaces were numerous solid cords of tumor cells the structure of which was exactly similar to those lining the blood spaces. They were composed of oval or indented vesicular nuclei around which was an indistinct cytoplasm. Supporting the blood spaces was a thin, delicate connective tissue stroma. In some parts of the tumor this connective tissue became very abundant and was found in broad sheets. Here and there small spicules of bone were found without evidence of reaction within them. Reticulin was present as a delicate framework throughout the tumor.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1. Sagittal view of the thoracic spine showing by stereoscope only the characteristic appearance of the body of the eighth vertebra upon which the diagnosis was based. This view shows a soft tissue shadow mainly to the left of this vertebra which was very confusing and which led to a suspicion of a cold abscess or large tumor such as sarcoma. The final identity of this shadow, however, was never determined.

Fig. 2. Lateral view of the thoracic spine, showing the characteristic appearance of the body of the eighth vertebra which suggested the final diagnosis of hæmangioma. The mottled appearance of this vertebra extends posterior into the lamina.

Fig. 3. Sagittal roentgenogram of thoracic spine after injection of campiodol (1.25 cubic centimeters) into spinal fluid, needle being inserted between the third and fourth lumbar vertebræ. With the patient in the inverted position the campiodol shadow is seen to be stopped at the level of the intervertebral space between the eighth and ninth thoracic vertebræ, indicating a block at this point. The metal identification marker is at the level of the body of the ninth vertebra.

A recent review of cavernous hæmangioma of the vertebræ by Bailey and Bucy included 11 cases of this type of lesion which had caused cord compression, and none of which were diagnosed correctly before operation. Four of them were operated upon and 2 survived, 1 of them being the case reported by Bailey and Bucy.

Our case presents several points of interest which makes it worthy of record. The condition was correctly diagnosed from the roentgenograms and the patient made a complete recovery following operation.

The roentgenologic features of hæmangioma of the vertebræ consist, according to Bailey and Bucy, of "reduction in bone density between parallel vertical trabeculæ which are increased in density." This, they regarded as pathognomonic of angioma of the vertebræ. It was due to the signalizing of these features by these authors, that a roentgenologic diagnosis of angioma of the vertebræ was made by one of us. It will be seen from the accompanying reproductions from the roentgenograms that the features as pointed out by Bailey and Bucy are well exemplified in our case. To the description of these authors we would add that there is an appearance of large vacuoles due to the presence of large vascular channels in the bone, and which do not assume the locations or take the known courses of the normal vessels. This whole appearance is analogous or in a way similar to that found associated with cavernous hæmangiomata of the skull, described by Cushing and more in detail by Sosman as a typical honeycomb or network appearance. The reasons for these appearances in the vertebræ and the cranial bones are obvious.

Despite the roentgenological diagnosis in the case here reported there was some hesitancy about its acceptance clinically, because of the fact that the condition is as yet so little known and so rarely encountered. The diagnosis was fully confirmed, however, both at operation and histologically.

From the operative standpoint, our case represents the third which has been successfully treated surgically. The patient showed prompt improvement following operation, and 3 months later had completely recovered the power in her legs, and was entirely well.

There are some features of the clinical aspects of these tumors which deserve mention. Not all angiomata of the vertebræ cause compression of

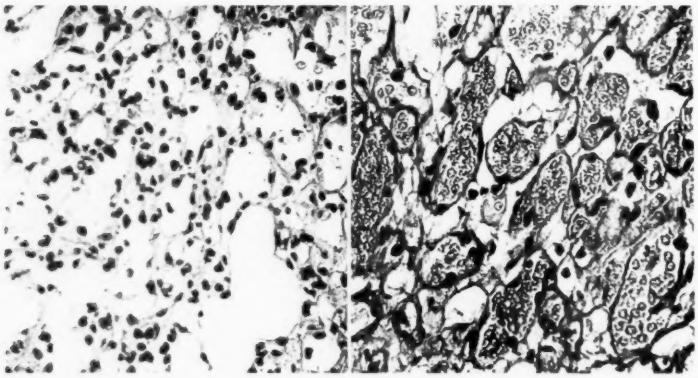

Fig. 4. Left, low power view of the tumor showing the numerous capillary and cavernous blood spaces lined with endothelium. Solid columns of endothelial cells are seen here and there in the tumor. Right, higher power view showing many of the spaces filled with blood.

the spinal cord. Those which do so produce their effects probably by extension of the tumor from the vertebra to the extradural area, thus causing cord compression without much narrowing of the lumen of the vertebral canal. In our case the latter was not narrowed to any appreciable extent as far as the naked eye could observe, but there was a definite extension of the tumor along the outer side of the dura. Incision of the latter showed no tumor tissue to be present subdurally. In the case of Bailey and Bucy, there was not only a marked narrowing of the spinal canal but "the peridural tissue was found to be exceedingly vascular."

Furthermore, sensory levels in these cases seem to be notably vague and indefinite. This was so in our case, and seems to have been true of the other cases collected by Bailey and Bucy.

At one time a complete subarachnoid block was demonstrated in our case by Queckenstedt test but could not be confirmed on a second occasion. This suggests the possibility that there may be a certain amount of expansion of these tumors due to filling of the blood spaces, and that at such times the symptoms may be definitely aggravated. The course of our case would tend to confirm this view because there were periods during the course of her illness in which she lost some of h symptoms and seemed to be almost in a remissio. It is our belief that an increase and decrease in blood flow through the tumor, expanding and collapsing the cavernous and capillary spaces, may account for the variations in symptoms, and for the appearance and disappearance of subarachnoid block.

CONCLUSIONS

1. A case of hæmangioma of the vertebra is reported.
2. A pre-operative diagnosis was made by means of the characteristic roentgenographic appearance.
3. Operation was successful, and the patient made a complete recovery.

BIBLIOGRAPHY

1. Bailey, P., and Bucy, P. C. Cavernous hæmangioma of the vertebræ. J. Am. M. Ass., 1929, xcii, 1748.
2. Cushing, H. Surgical end results in general with a case of cavernous hemangioma of the skull in particular. Surg., Gynec. & Obst., 1923, xxxvi, 303–308.
3. Sosman, M. C. Radiology as an aid in the diagnosis of skull and intracranial lesions. Radiology, 1927, ix, 396–407.
4. Spiller, W. G. Personal communication.

FRACTURES OF THE WRIST

A Review of One Hundred Seventy-Six Cases

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A REVIEW has been made of all fractures about the wrist seen at The Mayo Clinic in the period of 5 years ending July, 1930. We have included all "old" fractures, whether or not they were treated, and all "fresh," or acute, fractures treated. We have regarded as fresh fractures all of those seen within a period of 2 weeks of the inciting injury. The two groups offer an interesting parallel study and emphasize certain facts which would not be brought out in a study of either group alone. The series consists of 176 fractures; 87 fresh, and 89 old. As part of this study, we have gathered data on end-results concerning cases of acute fracture, so far as possible, and concerning cases of old fracture, if operative treatment was carried out. These studies of end-results are tabulated. In the majority of cases the roentgenograms have been reviewed and diagnosis verified. So far as possible pre-operative as well as postoperative roentgenograms were reviewed.

FRESH FRACTURES

In our grouping (Table I) we have not included all of the names sometimes used to designate these fractures, such as "Smith's," "Barton's," and so forth. But for its common usage and historic importance we might have omitted the term "Colles'" fracture and have substituted the term "suprastyloid," as suggested by Eliason. Actually, as has been frequently pointed out, Colles did not describe what is now commonly known as a Colles' fracture, but a fracture of both bones higher in the forearm. According to Davis, Colles, in his description published in 1814, placed the site of fracture 1½ inches (4 centimeters) above the joint. The same author stated that the fracture had been described previously by Pouteau and Nélaton, in 1783, but that it was "largely due to Robert W. Smith's *Treatise on Fractures in the Vicinity of Joints*, Dublin, 1847, that the name 'Colles' has become generally accepted." Smith placed the fracture a quarter of an inch to 1 inch (0.5 centimeter to 2 centimeters) above the joint and this situation has been generally accepted by all authors since that time.

Colles' fractures. In classifying the Colles' fractures we have purposely omitted the term "impaction." In our experience, in the majority of these cases there is some impaction when the cases are first seen. The nature of the bone involved, and the mechanism of the fracture, make this usual. We have grouped these as comminuted or not comminuted, which seems to us far more important. This complication at once leads to modification of the prognosis and occasionally to modification of treatment. No doubt such a fracture as that known as Barton's does occur and was present in some of these cases, but to us its treatment and prognosis do not seem sufficiently distinctive to warrant separate classifications.

The most important point to note in the original roentgenogram, then, is the presence or absence of comminution. With comminution, particularly if it extends into the joint, breaking the articular surface, the prognosis at once must be a little more guarded than otherwise. Any injury to the articulating surface of the radius is bound to set up active traumatic arthritis in the radiocarpal joint. Comminution occurred in slightly less than half of our cases. It may have been present in a few of those in which it was not noted, and in which we were unable to review the original roentgenograms. Its presence in 50 per cent of the cases is, however, of much significance.

Fracture of the styloid process of the ulna is a second point of importance to note. In this series, it occurred in approximately 63 per cent of the cases. It should be noted for two reasons. First, no doubt it makes accurate reduction more difficult. Second, in many cases in which it is fractured, tenderness and swelling about it are the most persistent signs of disability. Other authors have reported that this complication is seen in from 40 to 50 per cent of Colles' fractures. Why the proportion should be higher in this series is not easily explained. There doubtless are many cases in which actual fracture of the styloid process of the ulna cannot be demonstrated in the roentgenogram, but a tear of the ulnar collateral ligament takes place. Injury to the distal radio-ulnar

joint must occur in many of these cases. Often definite separation or comminution of the ulnar border of the articulating surface of the radius is seen in the roentgenograms. All of these facts should be remembered and if it is possible to visualize the lesions, they should be noted. We have frequently noticed swelling and effusion along the flexor tendons. This would be expected, and in our experience may nearly always be noted. It is apparently usually of no significance, but it may cause rather prolonged limitation of motion in the flexor tendons, and is particularly troublesome if patients are loath to use their fingers early in the treatment. It is no doubt a very important contributing factor in cases in which prolonged immobilization leads to prolonged disability.

Injury to the nerves in our series of cases was not common. Turner noted that injury to the ulnar nerve may occur, as evidenced by excessive perspiration and hyperæsthesia over the palmar side of the carpal bones, hyperæsthesia of the thenar eminence, and pain over the distribution of the ulnar nerve, particularly if the ulnar styloid process is torn off. According to the same author, the most serious change occurs in the dorsal interosseous nerve. He stated that this is evidenced by œdema distal to the second phalanges of all four fingers, glossy and reddened skin, stiffness of the fingers, with limitation of active and passive motion, and osteoporosis. Exploration in these cases revealed a spindle shaped and congested nerve. These complications may be present more frequently than we have noted. We have not found them and we doubt their serious importance.

Fractures other than Colles'. Fracture of the styloid process of the radius is seen more often in backfire fractures. It is less often displaced than any of the other fractures of the wrist. In two of our cases it was accompanied by fracture of the styloid process of the ulna, whereas, in another case, fracture of the navicular and capitate bones was seen complicating fracture of the styloid process of the radius.

Epiphyseal separations represent about 13 per cent of all the fresh fractures at the wrist. These are essentially the Colles' fractures of children, although typical Colles' fractures, too, may be seen in children. Greenstick fractures, and fractures of both bones of the forearm, near the wrist, are more often seen in children than in adults. An occasional subperiosteal fracture, near the joint, may be seen in adults, although it is a comparative rarity. In this group of acute fractures of the wrist, the navicular was the only carpal bone of which fracture was noted. This is the carpal bone most commonly fractured, according to all series thus far published. No acute injuries to the semilunar bone were noted during this period.

TABLE I.—FRESH OR ACUTE FRACTURES

Both radius and ulna		
Colles'		55
Comminuted, with fracture of styloid process of ulna	19	
Comminuted, without fracture of styloid process of ulna	7	
Not comminuted, with fracture of styloid process of ulna	15	
Not comminuted, without fracture of styloid process of ulna	14	
Reverse Colles' (with fracture of navicular)		1
Styloid process of radius (two with fracture of styloid process of ulna also; one with fracture of navicular and capitate)		8
Epiphyseal separations		11
Greenstick and subperiosteal		5
Both radius and ulna near wrist		3
Carpal bones		
Scaphoid		4
Total		87

Treatment of fresh fractures. The treatment of acute Colles' fractures has been described adequately many times. It may be summarized in three expressions; accurate reduction, adequate fixation, and early motion. If each of these requisites is followed, there will be few failures in the treatment of this injury. That many still are unsatisfactorily treated is sufficiently emphasized by the fact that in the period covered by this study we saw many cases of old Colles' fracture with unsatisfactory results.

In the first place, reduction must be accomplished. It is a very rare Colles' fracture that does not need some manipulation if uniformly good results are to be obtained. It is extremely rare, and perhaps impossible, to have a Colles' fracture in which careful examination of the wrist and roentgenogram will not reveal some deformity. Slight deformities may be overlooked at the time of treatment, but they will not be overlooked by the patient later on. There undoubtedly are occasions and circumstances in which manipulation is contra-indicated, but we emphasize the fact that they are rare. In cases of fracture of the styloid of the radius, without displacement, manipulation is not needed. Greenstick and subperiosteal fractures can go without manipulation if there is no deformity or if the deformity is very slight.

The choice of anæsthetic is important because anæsthesia usually is necessary. Many advocates of local anæsthesia are found today. If in their hands it is successful and their results justify its use, well and good. Our choice is general anæsthesia. We have not had any trouble with it, and

TABLE II.—END-RESULTS IN ACUTE OR FRESH FRACTURE

Types of fracture	Total number of cases	Cases followed	Result			Cases not followed
			Good	Fair	Poor	
Comminuted Colles' with fracture of styloid process of ulna	19	14	11	3	0	5
Comminuted Colles' without fracture of styloid process of ulna	7	6	4	0	2	1
Colles' with fracture of styloid process of ulna	15	10	9	0	1	5
Colles' without fracture of styloid process of ulna	14	9	6	3	0	5
Styloid process of radius	8	8	6	2*	0	0
Epiphyseal separation	11	6	6	0	0	5
Greenstick and subperiosteal	5	2	2	0	0	3
Radius and ulna near wrist	3	2	0	2 (1 compound)	0	1
Navicular	4	3	3	0	0	1
Reverse Colles'	1†	1	0	1	0	0
Total	87	61	47	11	3	26
All cases (per cent of 87)			54	13	3	30
Cases followed (per cent of 61)			77	18	5	

*Styloid of radius, scaphoid and dislocated capitate.
†Comminuted, compound fracture of navicular and fifth metacarpal.

see no reason for having trouble with the short anæsthesia by nitrous oxide gas and oxygen, or ethylene, necessary to accomplish reduction, particularly if skilled anæsthetists are available.

The type of fixation is worthy of some comment. Our choice is two splints of plaster of Paris, one on the dorsal aspect, running from the metacarpophalangeal joints to just below the elbow, the other on the palmar or volar aspect, running from the palm, at the metacarpophalangeal joints, to a point far enough below the elbow to allow free flexion of that joint. These should be applied with the hand pronated and the wrist palmar flexed, in some ulnar deviation. Properly applied splints allow free motion of the fingers, which should be encouraged at once, and the rolled edge of the palmar splint makes a good grip on which to exercise the fingers. In this way firm fixation of the wrist may be obtained without loss of movements of the finger. This is certainly the best position in which to maintain complete reduction of a Colles' fracture. In many cases, no doubt, it may be maintained with the wrist straight. Very little padding should be used between the skin and the plaster splints. If the splints are carefully molded while the plaster is setting and held in place by gauze bandages no trouble will be encountered. The patient should be warned of the danger of swelling and should be seen at least once in 24 hours after reduction. It will usually be necessary to cut the bandage and reapply it, although occasionally the swelling is not enough to demand this. Cotton has advocated leaving the wrist in this position for 3 weeks. In our experience this is not necessary in the majority of cases. Seven to 10 days suffice to get firm enough fixation so that the flexed position can be abandoned for a straight or slightly dorsiflexed position, maintained preferably on an aluminum cock-up splint. For older persons, in whose cases comminution is marked, longer fixation is necessary. It must be borne in mind, however, that, in these cases of older persons, trouble from too long fixation in the flexed position may result.

With the change of position and of splint, active movement may be commenced. If physiotherapy is available, and if the patient can afford it, restoration can be hastened by daily baking, light massage, and active exercises. A few instructions to the patient for home use will help very much. Most patients can be trusted not to overdo active movements. They are the most essential part of the program of rehabilitation, and no patient need omit daily active movement after the original fixation splint is removed. Roentgenograms for re-examination should be taken at the end of 10 days or 2 weeks. If any loss of position has occurred it usually is possible to correct it at this time, whereas later it may be impossible without refracture or osteotomy. In treatment of younger persons, all splints can be abandoned in 3 to 4 weeks; for older persons 5 to 6 weeks should be all that is necessary.

In fractures of the styloid process of the radius, without displacement, a splint should be applied for relief of pain. Early, active movements should be possible within a week, and the splint should be abandoned in 2 to 3 weeks at the most.

Epiphyseal separation should be reduced at once, under anæsthesia. Splinting of the same type as that just described should be applied, and movement should be started in one week. All splints should be abandoned in 2 weeks. Greenstick and subperiosteal fractures should be treated approximately according to the same schedule.

Fracture of both bones near the wrist is an entirely different problem if displacement occurs. Often the only method of getting satisfactory reduction of these fractures is by open reduction,

after which splinting in a straight position, either with plaster or with plaster on board splints should be carried out. Such splints should be left on 2 to 3 weeks before movement is started, depending on the age of the patient and the type of internal fixation, if any, that is used.

In fractures of the navicular, reduction can be accomplished by manipulation and the fragments are best held in position by dorsal flexion with some radial deviation of the wrist. In cases in which fracture of the navicular is complicated by fracture of the styloid process of the radius, or by a Colles' fracture one must choose between accurate fixation of the radius or of the navicular. The former is probably more important, and the position of choice would be the straight or slightly palmar flexed wrist. It is apparently generally agreed by all authors that prolonged fixation of 4 to 6 weeks at least is necessary in fractures of the navicular.

Follow-up study. Any method of follow-up study is at best not perfect. Ours has been carried on, in so far as possible, by personal examination and by letter of inquiry. Response has been at least as good as the average, as Table II will show. We have stated results as good, fair, and poor. Results were considered to be good if deformity was minimal or absent, and if there was normal range of motion, without pain. Results were graded fair if deformity existed which bothered the patient, and if motion was limited, with or without pain. Results were considered to be poor if there was decided deformity, with or without limitation of motion, and with or without pain. Personal examination is by far the most satisfactory type of follow-up.

We would like to call attention to the fact that the percentage of good results was higher in the more severe types of Colles' fracture, that is, those with fracture of the styloid process of the ulna. This probably means that reduction was carried out, and not simply splinting without reduction, which is always the temptation if slight deformity occurs.

OLD FRACTURES

This group, as we already have stated, includes all cases in which the injuries had existed 2 weeks or more. The time since injury ranged from 2 weeks to 7 years. Many of the patients were advised to have treatment, if necessary; many were reassured; some came obviously trying to make trouble for some physician with whose results they felt dissatisfied, but in most instances the poor results were due to the patient's own lack of co-operation. Studies of end-results were carried out only in those cases in which surgical procedures were adopted at the clinic (Table III).

TABLE III.—CASES OF OLD INJURY TO THE WRIST (MORE THAN TWO WEEKS SINCE THE INJURY)

Radius and ulna		
Colles' fractures		56
With fracture of styloid process of ulna (one bilateral)	29	
Simple Colles'	15	
Comminuted Colles' with fracture of styloid process of ulna (in three cases there was associated fracture of the navicular)	12	
Reverse Colles' fracture		2
Fracture of styloid process of radius		4
Fracture of styloid process of ulna		1
Subperiosteal fracture		1
Fracture of radius and ulna bones just above wrist		1
Carpal bones		
Injury to navicular (two bilateral, one dislocated)		16
Injury to lunate (three with fracture of styloid process of radius; three dislocated; three fractured)		6
Fracture of greater multangular (one with fracture of base of first metacarpal)		2
Total		89

Colles' fractures. In studying the cases of old Colles' fractures it was attempted to discover, in so far as possible, the causes of complaint which brought the patients to consultation. Deformity with or without pain was noted in 30 cases; stiffness of less than 2 months' duration in 6 cases; stiffness of more than 2 months' duration in 15 cases, and, concerning 5 cases the complaint was not stated. Deformity and stiffness were the most important complaints. Pain was nearly always present to some degree in the presence of one or another of these complaints. The reason for deformity is obviously inadequate reduction, and when one notes that anæsthesia was used for reduction of only 3 of these 56 fractures the answer is plain. Stiffness, in cases of less than 2 months' duration, may readily work out to a satisfactory end-result. In other cases it is almost invariably due to too long fixation in plaster or splint. In some cases it is no doubt due to lack of co-operation on the part of the patient. In one instance a large damage suit was pending, and the case hinged largely on the stiffness. How could the joint be otherwise than stiff? The importance of physiotherapy can be emphasized here again; it was the only treatment recommended in most of these cases and had obviously been neglected in many. The fact is worthy of note that the non-comminuted fracture predominates in numbers in this series. This is probably due to the fact that these do not appear at first to be so severe, and the temptation to let them go without reduction is often the reason for an inadequate reduction and therefore an unsatisfactory result.

TABLE IV.—OPERATIVE TREATMENT IN OLD CASES OF INJURY TO THE WRIST

Fracture	Operation	Cases	Result
Colles' type	Osteotomy	4	Good 3 Fair 1
	Excision of distal end of ulna	2	Good 1 Undetermined 1
	Capsulotomy of fingers for stiffness	1	Good 1
Of navicular	Excision of proximal fragment	3	Good 3
	Excision of entire bone	1	Good 1
Of lunate	Excision	4	Good 2 Fair 1 Poor 1
Of greater multangular	Excision	1	Fair 1

Operative treatment of old fractures. A summary of those cases of old injury in which operation was performed here reveals the facts recorded in Table IV. In all other cases of injury to the carpal bones, physiotherapy was recommended. In one case there was septic infection and operation was contra-indicated. The results in the cases in which operation was performed are certainly good enough to justify more extensive trial of such a procedure. Osteotomy of the lower end of the radius, with or without the use of a bone graft to hold the fragments in their new position, when carefully done, can greatly improve the appearance of a deformed wrist. Excision of the lower end of the ulna, if it is prominent, and if the lower end projects downward beyond the line of the radiocarpal articulation is a simple procedure worthy of further trial. It not only improves the appearance of the wrist but often improves the function greatly. Excision of the proximal fragment of the navicular is fairly well established, as is excision of the lunate bone.

Backfire fractures. There has been a great deal of attention paid to this type of fracture in the past 15 years. The importance of its mechanism of production seems to us overemphasized. We found 19 fractures produced in this way: styloid process of radius, 6; Colles' with fracture of ulnar styloid, 6; epiphyseal separation, 4; subperiosteal, 1; and 2 of which the type was not noted. Fracture of the styloid of the radius is relatively higher in frequency in this group; 6 out of 8 of such fractures were produced in this manner. Edwards has elaborated on the mechanism and classification of these fractures. Other authors stress their importance. Our feeling is that with the passing of hand cranking automobiles they are probably going to be fewer in number, and consequently of less importance.

SUMMARY AND CONCLUSIONS

We have tabulated a group of fresh or acute fractures, together with a group of old fractures about the wrist. The importance of accurate reduction has been emphasized, together with early motion. Studies of end-results in acute cases, as well as study of old cases shows a relatively higher percentage of poor results in the simpler types of fracture, probably because the tendency is to let them go without reduction under anæsthesia. We have tabulated a small group of results in old cases treated by operation, and can recommend further use of these operative procedures.

BIBLIOGRAPHY

1. Cotton, F. J. Adequate reduction and care in Colles' fracture. New methods. Boston M. & S. J., 1919, clxxxi, 651–656.
2. Davis, G. G. Applied anatomy. 6th ed. Philadelphia: J. B. Lippincott Company, 1924.
3. Edwards, H. C. The mechanism and treatment of backfire fracture. J. Bone & Joint Surg., 1926, xxiv, 701–717.
4. Eliason, E. L. Fractures of the humerus, radius, and ulna. New York: D. Appleton and Company, 1925.
5. Turner, H. Ueber Nervenschaedigunger beim typischen Radiusbruch. Arch. f. klin. Chir., 1924, cxxviii, 422–436.

EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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SEPTEMBER, 1932

THE IMPORTANCE OF CLINICAL DIAGNOSIS IN SURGICAL CONDITIONS OF ABDOMEN

IN the early days of surgical operations in the abdomen, methods of diagnosis had not been brought to a high pitch of accuracy. Even when an operative incision was made and the condition was under the eye, many times it was difficult to make a diagnosis which satisfied us that the condition which we had diagnosed, and perhaps had found, was sufficient to account for the patient's symptoms. Therefore, it was often almost a necessity at operation to make extensive examinations of all the abdominal viscera and sometimes to perform operations on organs other than the one primarily concerned.

In the course of time, as experience improved our diagnostic methods and gave us a clearer understanding of the causes of disease, we were better able to recognize disease conditions found at operation and relate them to the patient's symptoms, and knew that cure or relief would result from surgical intervention. We could not quite rid ourselves, however, of the habit of performing secondary operations for conditions of relatively little importance of which the patient had not complained. For example, when hysterectomy is performed for fibromyoma, the appendix is removed for cause, but formerly it was not unusual, if gall stones were found to be present, to remove them at the one operation; all quite proper, if the patient's condition warranted it.

But with increased experience, we grew more and more cautious about performing operations of doubtful necessity when operating for serious conditions, unless the state of the patient rendered it safe. We were careful to examine the whole abdomen with the gloved hand for evidences of pathological conditions, but we were increasingly conservative in operating as a routine for unimportant abnormalities.

When adequate pathological change is found to explain the symptoms for which the patient comes for relief, the making of larger incisions and more extensive explorations in the presence of a serious major condition, either because of the existing disease or because the patient is in poor condition, is not wise.

In my operative work, if I made no general abdominal exploration, I always told the patient and the family, and gave the reason why. If it became necessary later to remove a diseased appendix, the patient and his family understood the matter. Again, occasionally finding gall stones which probably would give trouble, I explained in each case to the patient and the family that it might be wise to remove the stones when convalescence from the immediate operation was well advanced and while the patient was still in the hospital.

At least I took pains that the persons concerned should be informed that gall stones were present, or that the appendix was or was not removed. In no instance that I can recall was the patient dissatisfied after such an explanation had been made.

The value of giving information of this sort at the time of operation is evident in connection with a subsequent illness of the patient, perhaps many years later. If the patient knows, for instance, that the appendix was or was not removed, the knowledge often saves the consulting physician and the patient the inconvenience and perhaps dangerous delay of waiting for information. In other words, the patient should be informed regarding the whys and wherefores of those minor conditions which so frequently are found in connection with a major operation.

In this connection my whole experience leads me to believe that the clinical examination with the history of the patient is most important. The evidence given by the X-ray and other precise means of examination is so extraordinarily good that one sometimes leans too strongly on these aids. Recently a distinguished internist contrasted our present-day diagnostic methods with the old, showing that before the X-ray played so great a part in the diagnosis of disease of the gall bladder as it does today, the clinical diagnosis was 90 per cent correct. Since the X-ray has been used to make the diagnosis rather than to confirm it, diagnoses of gall bladder trouble are only 70 per cent correct, the disease being overlooked. This loss in accuracy applies also to diagnosis of ulcer of the posterior wall of the duodenum and of the stomach. The X-ray will be correct in about 95 per cent of cases of ulcer, but in at least 5 per cent it may be misleading. We should remember that the X-ray film gives a picture of the shadow of the object, and not a photograph of the object itself. In the gall bladder, for instance, papillomata and cholesterosis may not produce X-ray evidence that stands out, and yet the disability and dangers for the patient are the same as if stones existed.

The corollary of my argument would be that the surgeon should not pay too much attention to the spectators in the seats or to what they may be thinking, but should give his undivided attention to the patient and to the patient alone. In major operations for serious conditions, any additional procedures should be considered very carefully lest they unnecessarily jeopardize a patient already seriously ill. Again, the laboratories should be used for the purpose of aiding the clinical diagnosis, but should not supersede it.

W. J. Mayo.

LETHAL FACTORS IN INTESTINAL OBSTRUCTION

MUCH effort has been expended to determine the cause of death in acute intestinal obstruction with the ultimate hope that something might be found to aid in reducing the very high mortality incident to this disease. It is quite to be expected that such an intricate problem would call forth many theories, some of which border on the fantastic. Attention has been chiefly centered upon the content of the obstructed gut in an effort to explain the development and absorption of a toxin. Any theory based upon the absorption of pathological products developed within the obstructed bowel to date have fallen short of an adequate explanation of the symptomatology and pathology of the disease.

In recent years the subject has been approached from the standpoint of perverted physiology and alteration of the chemical balance of the body. Much experimental evidence has accumulated which shows the

importance of a loss of upper intestinal tract secretions. A complete drainage of the stomach will cause death. A complete drainage of the pancreatic juice will cause death. Complete drainage of the duodenum or upper jejunum will cause death. There is, therefore, abundant evidence that a loss of the upper gastro-intestinal tract secretions will result fatally. A loss of these secretions by jejunostomy causes changes in the blood chemistry similar to those found in high intestinal occlusion. The old idea that the content of an obstructed gut, when released into the normal gut below will produce death, has definitely been found to be erroneous. In fact, quite the opposite has been proved true. The life of animals may be prolonged by deliberately injecting the stagnated content from above an obstruction into the normal bowel below. Substances accumulate in the obstructed gut which are essential to life. Toxic substances, such as histamin, injected into an obstructed bowel are not absorbed. It, therefore, seems clear that toxic contents of an obstructed bowel are not absorbed, producing the so called toxæmia of intestinal obstruction.

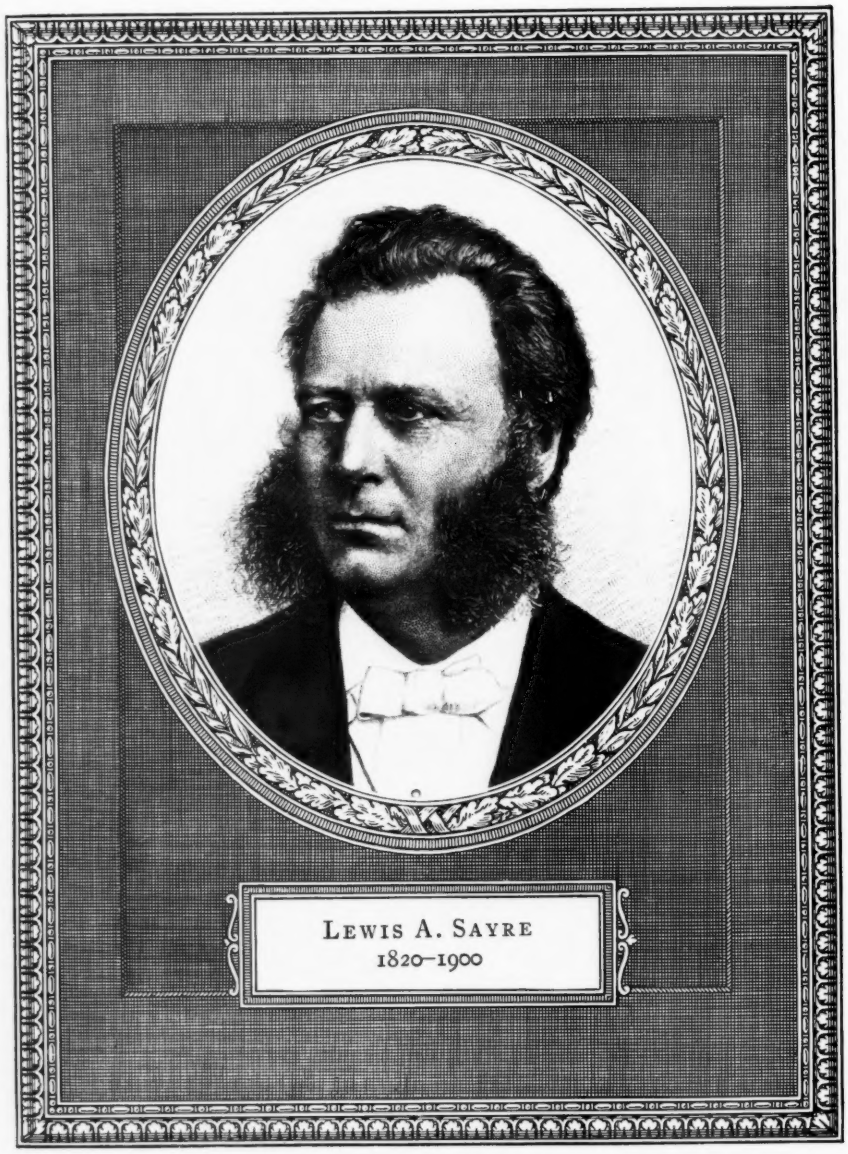
Typical changes in the blood chemistry occur in high bowel occlusion, the most striking of which is the diminution in chlorides. There is also a rise in the non-protein and urea nitrogen and a rise in the carbon dioxide combining power of the plasma. In addition to these easily demonstrated changes, there is an increase in the total protein, fibrin, viscosity, sedimentation rate, coagulability, and a decrease in the oxygen capacity. These changes indicate a marked disturbance of the physicochemical properties of the blood, probably very largely due to liquid loss.

Marked dehydration results from a loss of upper alimentary tract secretions. Dehydration, experimentally produced, causes an increase in the nitrogenous elements of the blood, indicating a disintegration of protein tissue. This change is not unlike that produced by obstructive lesions of the small bowel. It is possible that the increase in non-protein and urea nitrogen in the latter may be the result of a rapidly developing dehydration due to vomiting or accumulation of liquid in the stomach or obstructed bowel.

It is an interesting fact that life can be much prolonged and the changes in the chemistry of the blood prevented or restored to normal limits by the administration of a proper quantity of sodium chloride and water.

What then is, at present, the most reasonable explanation of the cause of death in simple obstruction of the small intestine? With the evidence as stated in mind, the only logical conclusion to be drawn is that the lethal factors are produced by a loss of secretions from the stomach and upper intestine which are essential to life, with a marked development of hypochloræmia and dehydration. This knowledge has quite naturally led to the replacement of sodium chloride and water which has become such an important phase of the treatment of obstructive lesions of the gastro-intestinal tract.

THOMAS G. ORR.



LEWIS A. SAYRE
1820–1900

UNIV. OF MICH.

MASTER SURGEONS OF AMERICA

LEWIS A. SAYRE

FROM the beginning down to this very day no one has influenced orthopedic surgery in this country as did Lewis Albert Sayre. And this, despite the fact that he wished to be known as a surgeon—not as an orthopedic surgeon. When the formation of the American Orthopedic Association was being debated, he opposed the plan and urged that all those interested apply for membership in the American Surgical Association, of which he was a member. He refused to join the new organization, although his sons, Lewis Hall Sayre and Reginald H. Sayre were charter members, and the former was secretary at the first meeting in 1887.

Like all really great men, Lewis A. Sayre bulked high from many points of view; which is much the same as saying that he had good friends and good enemies, and probably enjoyed his enemies as much, or more, than he enjoyed his friends. I think he may, at least for a time, have counted me among his enemies, but no one ever showed me greater personal kindness than did Doctor Sayre. It was always a proud day for me when he would drive up to my house in his open carriage, handsome pair of black horses, and coachman in livery, and shout at the top of his great voice: "Ho, Ridlon! Ho, Ridlon!" until someone heard him and called me to the door, and we would go for a drive. On one such occasion I asked: "To what one thing more than any other do you attribute your great success as a surgeon?" Without hesitating an instant he replied: "More to what I don't know than what I do know." I asked him to explain just what he meant. He said: "I have never been much of a reader, and so have not known what other men have tried to do and have failed in doing. If a problem presented, I did what I thought it best to do, and have usually succeeded. But if I had known that good men had tried to do the thing and had failed, I probably would have not made the attempt, or would have failed from lack of self-confidence." This self-estimate may well be kept in mind when considering the notable incidents throughout his whole life. It explains many things, and softens the sharp lines of our criticism.

Lewis Albert Sayre was born in Battle Hill (now Madison), Morris County, New Jersey, on February 29, 1820. He died in New York City on September 21, 1900. His father, Archibald Sayre, was a farmer. His grandfather, Ephraim

Sayre, was a quartermaster in the Revolutionary Army. After the death of his father, Lewis was sent as a boy of nine or ten years to Lexington, Kentucky, to be educated by his uncle, David A. Sayre, the principal banker of that town, who had no children of his own. His uncle was a pillar of the Presbyterian church and very desirous to have Lewis study for the ministry. But when the boy graduated from the Transylvania University (Lexington, Kentucky) in 1839 he insisted upon the study of medicine, came to New York, and with little help from Uncle David, graduated from the College of Physicians and Surgeons (New York) in 1842. He was prosector to the professor of surgery for about ten years. In 1853, he was appointed surgeon to Bellevue Hospital, and, in 1859, to Charity Hospital. He was one of the most active of the founders of Bellevue Hospital Medical College, and became professor of orthopedic surgery and fractures and luxations, later designated as that of orthopedic surgery. When the College was united with the New York University in 1898 he became emeritus professor of orthopedic surgery of the consolidated schools. He was one of the founders of the New York Academy of Medicine, the New York Pathological Society, and the American Medical Association, of which he was president in 1880.

It was in 1885 that the American Medical Association divorced from membership the New York State Medical Society for ethical infidelity (consulting with homeopaths) despite the valiant efforts of Dr. Sayre. At that time New York City had many eminent consultants while the rest of the country had relatively few; and the great men of New York refused to submit to the dictation of the Chicago group. Sayre was great enough to do as he liked; lesser men wished to abrogate a law that they were unwilling to obey.

In 1860, Fernando Wood was elected mayor of New York City for the third time. He summoned Dr. Sayre to his office and offered either of two medical positions at his disposal. Dr. Sayre reminded him that he was of the opposite political party, had opposed Wood's election, and would always oppose him. Wood replied that that had no bearing on the appointment. One position carried quite a large salary and only routine duties; the salary of the other was about half as much. Dr. Sayre chose the latter, that of resident physician to the City of New York, because it offered opportunity to attack sanitary evils. He continued to hold this position under the three succeeding mayors. He improved street-cleaning, the sewerage system, the small-pox situation, and tenement-house evils. He demonstrated the fact, not then accepted, that cholera is a contagious, not merely an epidemic, disease, and hence amenable to proper quarantine precautions, and demanded that the Federal Government be held responsible for the protection of the entire nation.

In 1871, Dr. Sayre visited Europe and received many honors; and he went again in 1877 as a delegate from the American Medical Association to the British

Medical Congress, and again received much honor. He is credited with the invention of the uvulatome, a club-foot shoe, a scrotal clamp, the flexible probe, an improved tracheotomy tube, a periosteal elevator, a suspension apparatus, the plaster-of-Paris jacket (and Jurymast) for the treatment of Pott's disease, a traction splint for hip disease, and splints for the treatment of other tuberculous joints. As to the "Sayre suspension apparatus," still appearing as such in surgical instrument makers' catalogues, he had a lengthy controversy as to priority with Dr. Benjamin Lee, of Philadelphia, and finally admitted Lee's priority, neither of them knowing that the same apparatus had been used by Hippocrates 400 years B.C. Doubtless both he and Lee devised the same apparatus. As to the traction splint for hip disease, Dr. Henry G. Davis originated the idea of elastic traction by a splint that permitted the patient to walk. Sayre made an improved splint. But today none of the apparatuses are in use as Dr. Sayre used them, This should in no way be construed as a reflection on their author; for not one surgeon in a thousand knows how properly to "set" a Pott's fracture, having forgotten, or never heard of the method recommended by Percival Pott two hundred years ago. Any surgeon who does one good new thing that remains a good thing for as long as twice his life-time is a rare man.

Dr. Sayre was the first to excise any considerable number of tuberculous hips. He did his first excision in 1854. For the ten year period 1859–1868, he excised 29; for the ten year period 1869–1878, he excised 40; for the ten year period 1879–1888, he excised 3. After that time, none. This illustrates the greatness of the man: To be able to learn from his own work *what not to do.*

When I last called on Dr. Sayre he was reclining on a couch in his dining-room, crippled with arthritis in both knees, while the boys, Lewis and Reggy, were caring for the patients in the office across the hall. He struggled to his feet and took two or three steps to reach the box of cigars, and then back again, meanwhile urging me never to get a carriage but to walk.

In 1849, he married Miss Eliza Ann Hall, daughter of Charles Henry Hall, of New York City, who had much to do with the development of that part of the city called Harlem. All her life Mrs. Sayre sincerely believed that her husband could do no wrong. To them were born Mary Hall, Charles H. H., Lewis Hall, and Reginald H. All the sons were graduates in medicine. Charles died (by accident) before I knew the family. Lewis and Reginald were eminent practitioners of orthopedic surgery. They were my friends. They are dead. Only Miss Mary remains. In writing this short sketch of her father, I owe much to her for personal letters, for a reprint from Leslie's *History of Greater New York*, and for the picture here printed. It shows Doctor Sayre in his lusty manhood—at his very best—a great man, with a great heart that held no drop of gall. It was all sweetness.

JOHN RIDLON.

REVIEWS OF NEW BOOKS

THE *Practitioners Library of Medicine and Surgery*[1] is a series of 12 books designed by its author, George Blumer, as a working library for the physician and surgeon. Dr. Blumer states that the man in general practice is deluged with periodical literature and reviews. "Text books grow larger and the systems of medicine being seldom revised, they quickly undergo a degenerative metamorphosis." The author feels that there is a need for a work covering the entire field of endeavor in practical medicine and surgery. ". . . great pains have been taken to include every essential, and by thorough indexing, make the material quickly available. The majority of the contributors are young men, selected because they are abreast of the rapid changes being made in the act of healing today." This material has been "arranged so that reference to a particular point is easy, and where bibliographies are given they contain references to outstanding articles."

The first volume contains 1271 pages of well printed material which serves as an introduction to the volumes to follow. The author states that the chief purpose of this volume is to put into the hands of the practitioner the means of orientating himself in the relationships which exist between the science and the art of medicine. The first 675 pages are used to present "anatomy as far as possible from the physiological standpoint, emphasizing the bearing of various anatomical observations on clinical medicine and surgery." For example, the first chapter of 50 pages is given to "Bodily Malformations in the Light of Embryology." The balance of the section is given to brief practical statements on general anatomy with excellent anatomic illustrations taken largely from good texts on anatomy.

The second half of volume i is given to similar physiology with "emphasis upon the points bearing on the actual practise of medicine." The first chapter of this section (Chapter viii) is given to "Heredity in Relation to Practical Medicine" and the author writes the next chapter on "General Considerations Concerning the Relation of Constitution to Disease." A fine chapter on "Energetics, Dietetics and Metabolism" together with sections on the circulation, digestion, liver, kidneys, endocrine glands, heat regulation and fever, and other interesting topics make up this volume.

Volume ii concerns itself with the technique of physical and laboratory examinations in clinical medicine. The editor writes the first chapter of 20 pages on history taking and the technique of diagnosis. This should be read by every medical student and used by the interne. This chapter is followed by 43 pages on physical diagnosis. A historical note accompanies each topic in the chapter on "Methods and Instruments of Precision in Diagnosis." The section of 160 pages on the blood by Thomas E. Buckman is well arranged and it is full enough to serve as a practical manual. The illustrations are mostly new ones by Miss Piotti. Another small section on the blood appears much later in the volume. A section is given to practical technique of diagnostic pathological examinations. One hundred ninety pages are given to the technique of bacteriological and immunological examinations in the diagnosis of infectious diseases and allergy. Sturgis and Root write on the endocrinopathies and diseases of metabolism. A separate section is given to tracheoscopic and bronchoscopic study. Sections on history and examinations on all of the various organs or systems of the body comprise the remainder of the volume.

M. HERBERT BARKER.

THE two volumes of *Nouveau Traité de Pathologie Chirurgicale*[2] written by J. Maisonnet constitute "tome ii" of this extensive work. Part 1 deals with the lesions of the upper extremities: fractures, dislocations, traumatic lesions of the joints, deformities, neoplasms as well as lesions of the blood vessels and nerves. The work impresses me as a rather ambitious undertaking, splendidly done. Particularly well written up and illustrated are the discussions on fractures and dislocations. In this respect the work is as complete and as thorough as any recent publication dealing with that subject alone. There is perhaps an advantage from a didactic viewpoint not to limit the work to fractures and dislocations alone, but to include all of the lesions. It tends to make the work more comprehensive. This volume contains 685 pages of text and 853 illustrations.

Part 2 is devoted to the lower extremities and follows essentially the same scheme as Part 1.

The work should make a particular appeal to the surgeon interested in traumatic surgery as well as to the orthopedist. To the general surgeon and the general practitioner the work could well serve as a reference text.

GEORGE HALPERIN.

AT the Accident Hospital in Vienna about six years ago, Boehler introduced the so called non-padded plaster-of-Paris cast in the treatment of fractures. This cast was applied for fractures and dislocations which had been reduced under local anæsthesia and according to the method popularized by him. Dr. Schnek, the author of the book which is herewith reviewed,[3] has been first assistant in this work for several years, and this book is a comple-

[1] THE PRACTITIONERS LIBRARY OF MEDICINE AND SURGERY. Vol. i, Anatomy and Physiology as Applied to Practical Medicine. Vol. ii, The Technic of Physical and Laboratory Examination in Clinical Medicine. New York and London: D. Appleton and Company, 1932.

[2] NOUVEAU TRAITÉ DE PATHOLOGIE CHIRURGICALE. Published under the direction of A. Basset, H. Costantini, G. Jeanneney, J. Maisonnet, G. Miginiac. Vol. II, 1st and 2d part, Membres, by J. Maisonnet. Paris: G. Doin et Cie, 1931.

[3] THE TECHNIQUE OF THE NON-PADDED PLASTER CAST. By Fritz Schnek, M.D. With a preface by Lorenz Böhler, M.D. Authorized English translation by Douglas D. Toffelmier, M.D. Vienna: Wilhelm Maudrich, 1932.

mentary one to Boehler's book on the treatment of fractures. Schnek realizes that many men, however dexterous in routine fracture and orthopedic work, have failed in the proper application of the non-padded cast. It is for this reason that he has outlined the description and illustrated the method from beginning to end. The book is translated from the German by a man who has spent 2 years under the personal direction of Schnek.

In the Preface, Boehler emphasizes two constant requirements in the treatment of any fracture, i.e., good reduction and uninterrupted fixation until consolidation occurs. Schnek enumerates the following factors in the cast: fixation of a portion of the body; maintenance of a definite position of part of the body or extremities; elimination of muscle pull; protection of the soft parts. Schnek describes in detail the preparation of the bandages; the use of the plaster splint, and reinforcement or "reverse." He emphasizes the danger of circulatory disturbances produced by plaster of Paris and considers the surgeon responsible for the patient from the time the cast is applied until it is removed. He cautions nurses and internes to be everlastingly on guard for signs and symptoms of pressure such as changes in color, swelling, mobility, disturbed sensation, and pain. When untoward signs arise, the cast must be split or removed.

When a window is cut in a cast it should be replaced in order to prevent swelling which the author calls "window œdema." A perfectly fitting cast never causes pain. Schnek warns against allowing the cast to set while resting on a hard surface because the weight causes an indentation which will result in pressure.

All fractures should be checked by roentgenograms made immediately or soon after reduction. When roentgenograms must penetrate the cast, the dosage should be increased 10 per cent and the exposure 50 per cent.

In answer to the frequent criticism concerning the hair, he states that the hair dies in about 3 weeks and pulls out easily. When the cast is removed the skin is covered by a new growth which is not adherent to the cast.

All correction of position of the limb should be accomplished before the plaster has set to avoid the formation of wrinkles on the inside.

The following few notes will illustrate the author's attention to detail: (1) the dorsum of the toes must remain uncovered to allow free movement and observations for circulatory changes; (2) the knee joint should always be fixed in extension to prevent atrophy of the quadriceps; (3) fractures should not be immobilized in an overcorrected position; (4) the operator himself should hold the leg while the assistant applies the cast; (5) in hand casts there must be free extension and abduction of the thumb, and complete flexion at the fifth metacarpal phalangeal joint; (6) the proximal finger joint should never be held in overextension; (7) the aeroplane splint of the stock variety or of Cramer wire is preferable to plaster cast to immobilize the shoulder.

Schnek believes that Volkmann's ischæmic palsy is due to compression of the brachial artery and vein by the lower jagged edge of the proximal fragment in a supracondylar fracture near the elbow. He advises reduction with longitudinal traction on the humerus with the elbow at right angle. He discusses the proper position of the retention in cases of flat-foot.

The illustrations are well chosen and executed. The reader must be charitable in reading the translation. Regardless of whether the surgeon employs the non-padded cast or not, there is so much good information in this book that it is well worth reading. Every general, industrial, and, orthopedic surgeon should read this book. PHILIP LEWIN.

THE presentation[1] by Schmorl and Junghanns of the pathological and roentgenological anatomy of diseases, deformities, and anomalies of the spinal column will take first rank in the literature pertaining to this subject. The observations and opinions set forth are based on the findings in 10,000 spinal columns removed at necropsy, and the material is presented in such a manner as to leave no doubt concerning its scientific merits. Throughout the text the illustrations of gross sections of the spine are accompanied by roentgenograms of the same specimens. The illustrations are admirably reproduced with a uniform clarity which leaves little doubt as to the findings in each case.

In the discussion of the development of the vertebral body, Schmorl brings out some new facts particularly concerning his conception of the "so called" vertebral epiphysis. He emphasizes that unlike other epiphyses they do not add length to the vertebral growth and therefore should not be called epiphyses. These really develop in the intervertebral discs and later fuse with the vertebral bodies. Their presence as related to disease or trauma is frequently referred to in the text.

In the discussion concerning the intervertebral discs there is presented a comprehensive summary of Schmorl's ideas regarding the nucleus pulposus and annulus fibrosis. These ideas have already gained widespread recognition but have not heretofore been so clearly and completely set forth. Variation of the nucleus pulposus in normal and pathological states is fully discussed. The author states that clinically their importance may not be great but from the standpoint of traumatic condition they should be recognized.

The section pertaining to lumbosacral lesions will be of great interest to those interested in the problem of "Backache." The author distinguishes true spondylolisthesis, where forward displacement results from a solution of continuity of the pedicles or laminæ, from pseudospondylolisthesis, where there may be a forward displacement due to an elongation

[1] FORTSCHRITTE AUF DEM GEBIETE DER RÖNTGENSTRAHLEN. Edited by Prof. Dr. Grashey. Sup. vol. 43. DIE GESUNDE UND KRANKE WIRBELSÄULE IM RÖNTGENBILD; PATHOLOGISCH-ANATOMISCHE UNTERSUCHUNGEN. By Geh. Med.-Rat Prof. Dr. Georg Schmorl and Dr. med. Herbert Junghanns. Leipzig, Germany: Georg Thieme, 1932.

of the pedicles without any break in the bony arch.

There is an excellent discussion of metastatic lesions of the spine, and many tables illustrating the distribution of the various lesions as regards the type of tumor, age, and sex are given. The consideration of primary benign and malignant tumors is somewhat brief and a consideration of bony changes as related to primary tumors of the spinal cord and meninges might have been presented in more detail. This is the only portion of the work which seems at all inadequate.

This monograph is unquestionably a most valuable contribution to the literature, and as a reference work on conditions affecting the spine, I know of no equal. A very complete and well arranged bibliography is appended. JOHN D. CAMP.

IN one[1] of his ten books on surgical pathology Hertzler describes the surgical pathology of the vulva, vagina, cervix, uterus, tubes, and ovaries. The style of the book is most informal and the subject matter and illustrations are all drawn from the author's vast personal experience. This makes for interesting reading but gives expression to personal views which are at times unorthodox and entirely at variance with accepted teaching. The author states that carcinoma of the cervix must be diagnosed clinically and not microscopically and that "it is extremely rare for the microscope to detect a malignant lesion which cannot be diagnosed in the clinic." He further throws doubt on the existence of a "precancerous lesion." The author was unfortunate in that his clientele is such that he has apparently seen but one early carcinoma of the cervix, for it is obvious that the best surgical results are to be obtained in early carcinoma of the cervix before the malignancy has spread to the neighboring tissues.

The repeated misuse of the word "tubercular" when "tuberculous" is meant might be criticized; tubercle bacillus infections result in tuberculous salpingitis or vaginitis, not in tubercular salpingitis or vaginitis. RALPH A. REIS.

[1]HERTZLER'S MONOGRAPHS ON SURGICAL PATHOLOGY. SURGICAL PATHOLOGY OF THE FEMALE GENERATIVE ORGANS. By Arthur E. Hertzler, M.D. Philadelphia, Montreal, and London: J. B. Lippincott Company, 1932.

IN a volume[2] of six hundred pages De Lee and Greenhill present a review of obstetrical and gynecological literature for the year 1931.

The senior editor, in the obstetrical division, has included reviews from all phases of obstetrics arranged under the following headings: pregnancy, labor, the puerperium, the newborn, and miscellaneous subjects. Numerous articles on the Aschheim-Zondek test for pregnancy in all of its applications and modifications are fully reviewed. The practicability and unquestioned value of this test is impressed upon one more than ever after perusing the rather abundant literature accumulated on this subject during the past year. It is undoubtedly the most important contribution that has been made in this field in recent years. The treatment of occipito-posterior positions, eclampsia, and placenta prævia is given considerable space, as is cæsarean section in the treatment of various complications of pregnancy and labor. The work of J. Whitridge Williams on the changes occurring at the placental site is thoroughly reviewed and illustrations are included. The original article is well worth the reading time of anyone interested in obstetrics. In dealing with the newborn, there seems to be a dearth of literature on birth-injuries as well as ways and means to prevent them.

Some very interesting abstracts are to be found under the miscellaneous section on maternal and child welfare, these being reviews of work on the subjects by Pollock, Holmes, Adair, and others.

The last three hundred pages of the volume contain a review of the gynecological literature in which the editor presents a very worth while lot of abstracts. The subject of sterility is given its rightful share of space and the importance of the Rubin test is properly emphasized by various authors. Ectopic pregnancy, operative technique, menstruation, and its disorders, infections, glands of internal secretion, tumors, benign and malignant, electro-therapy, and radiology are treated in the order given, each review presenting some interesting phase.

This volume is well worth having at hand as a reference to the past year's literature on these two subjects. CHESTER C. DOHERTY.

[2]THE PRACTICAL MEDICINE SERIES. OBSTETRICS. Edited by Joseph B. De Lee, A.M., M.D. GYNECOLOGY. Edited by J. P. Greenhill, B.S., M.D., F.A.C.S. Series 1931. Chicago: The Year Book Publishers, Inc., 1932.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

TWENTY-SECOND ANNUAL SESSION

ST. LOUIS, OCTOBER 17-21, 1932

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CLINICAL CONGRESS PROGRAM IN BRIEF

ALL SESSIONS AT JEFFERSON HOTEL EXCEPT AS NOTED

Monday, October 17

10:00. Hospital conference.
2:00. Clinics in hospitals.
2:00. Hospital conference.
2:00. Surgical film exhibition—Statler Hotel.
8:15. Presidential meeting.

Tuesday, October 18

9:00. Clinics in hospitals.
10:00. Hospital conference—Tuttle Memorial Auditorium.
10:00. Surgical film exhibition—Statler Hotel.
2:00. Clinics in hospitals.
2:00. Hospital conference—Tuttle Memorial Auditorium.
2:00. Surgical film exhibition—Statler Hotel.
8:00. Hospital conference—Tuttle Memorial Auditorium.
8:15. Scientific session, general surgery.
8:15. Section on ophthalmology—Statler Hotel.

Wednesday, October 19

9:00. Clinics in hospitals.
10:00. Hospital conference—Tuttle Memorial Auditorium.
10:00. Surgical film exhibition—Statler Hotel.
2:00. Clinics in hospitals.
2:00. Hospital conference—Tuttle Memorial Auditorium.
2:00. Surgical film exhibition—Statler Hotel.
2:30. Symposium—Treatment of Fractures.
2:30. Symposium—Teaching of Surgery.
8:00. Community Health Meeting—St. Louis University Gymnasium.
8:15. Scientific session, general surgery.

Thursday, October 20

9:00. Clinics in hospitals.
10:00. Hospital conference at hospitals.
10:00. Surgical film exhibition—Statler Hotel.
1:30. Annual meeting of College.
2:00. Clinics in hospitals. Symposium on cancer.
8:15. Scientific session, general surgery.
8:15. Section on otolaryngology—Statler Hotel.

Friday, October 21

9:00. Clinics in hospitals.
10:00. Surgical film exhibition—Statler Hotel.
11:00. Meeting of new Fellows, class of 1932.
2:00. Clinics in hospitals.
2:30. Conference on industrial medicine and traumatic surgery.
2:00. Surgical film exhibition—Statler Hotel.
8:15. Convocation.

AMERICAN COLLEGE OF SURGEONS

PRELIMINARY PROGRAM FOR THE ST. LOUIS CLINICAL CONGRESS

A PRELIMINARY program of the clinics and demonstrations to be given in the hospitals and medical schools of St. Louis during the twenty-second annual Clinical Congress of the American College of Surgeons, October 17–21, prepared under the supervision of the Committee on Arrangements, will be found in the following pages. The surgeons of St. Louis are planning to provide a complete showing of the surgical activities of their city, with its two splendid medical schools and many fine large hospitals, for the entertainment of the Fellows of the College and their guests. During the weeks preceding the Congress the hospital schedules are to be still further revised and amplified, so that in its final form, as it will appear in the October issue of SURGERY, GYNECOLOGY AND OBSTETRICS, the program will present a more completely detailed schedule of the clinical work to be demonstrated.

It will be noted that clinics are scheduled for the afternoon of Monday, October 17, beginning at 2 o'clock, and for the mornings and afternoons of each of the four following days and that the program includes operative clinics and demonstrations in all branches of surgery—general surgery, gynecology, obstetrics, orthopedics, urology, proctology, ophthalmology, otolaryngology, etc.

The clinical program as published at this time is merely an outline or basis for the final program. During the Congress the clinical program will be published daily in the form of bulletins prominently displayed on large bulletin boards at headquarters at the Jefferson Hotel. These bulletins will be posted each afternoon showing in complete detail the clinics to be given on the following day. The same material will be published in printed form in the *Daily Bulletin* and distributed to the visiting surgeons early each morning.

The clinical program presented by the St. Louis surgeons will provide many special features including (1) demonstrations of modern methods in the treatment of fractures at several of the hospitals where plans have been made for a comprehensive showing of the methods used and the results obtained in the treatment of fractures, which forms so large a part of surgical work in large cities and industrial centers; (2) demonstrations of the treatment of cancer by surgery, radium and X-ray; (3) rehabilitation by surgery and physiotherapy of patients injured in industrial and automobile accidents, etc.; (4) surgical research and experiment.

An extensive program of surgical film contributions will be presented including the newest films, both sound and silent, to be shown daily in the ballroom of the Statler Hotel.

Among the distinguished visitors from abroad who will attend the Clinical Congress and participate in its activities are Sir William I. DeCourcy Wheeler, past-president of the Royal College of Surgeons of Ireland; Sir George Lenthal Cheatle, consulting surgeon, King's College Hospital, London; Dr. José Goyanes, professor of surgery in the National Academy of Medicine of Madrid, Spain, and president of the Society of Surgeons of Madrid.

The annual meeting of the American College of Surgeons will be held in the ballroom of the Jefferson Hotel on Thursday afternoon, beginning at 1:30, for the reception of reports of officers and standing committees and for the election of officers, regents and governors.

EVENING MEETINGS

The Executive Committee of the Clinical Congress has prepared programs for a series of five evening meetings as presented in the following pages. At the presidential meeting on Monday evening in the ballroom of the Jefferson Hotel following the introduction of distinguished guests, the president-elect, Dr. J. Bentley Squier, of New York, will be inaugurated and deliver the annual address. This will be followed by the annual John B. Murphy oration in surgery delivered by Sir William I. DeCourcy Wheeler, of Dublin, Ireland. On Tuesday, Wednesday and Thursday evenings at meetings in the ballroom of the Jefferson Hotel papers on various surgical subjects of present-day interest will be presented and

discussed by a number of eminent surgeons of the United States, Canada and England.

Two meetings of special interest to ophthalmologists and otolaryngologists will be held in the ballroom of the Statler Hotel on Tuesday and Thursday evenings at which men of outstanding experience in these specialties will present papers.

The 1932 class of candidates for Fellowship in the College will be received at the annual convocation on Friday evening, on which occasion Dr. J. Bentley Squier, of New York, will deliver the president's address and Robert A. Millikan, director of the Norman Bridge Laboratory of Physics of the California Institute of Technology, will deliver the fellowship address.

Two special orations are included in the program: (1) the annual oration on fractures by Dr. Philip D. Wilson, of Harvard Medical School and the Massachusetts General Hospital; (2) an oration by Dr. Frederic A. Besley, Chairman of the Board on Industrial Medicine and Traumatic Surgery, dealing with the present and future activities of this department of the College work.

SYMPOSIUM: CANCER IS CURABLE

A clinical symposium that will emphasize the curability of cancer will be presented on Thursday afternoon at 2:30 in the ballroom of the Jefferson Hotel immediately following the annual meeting. Brief, definite summaries of five-year cancer cures will be presented by a group of clinicians who have made a special study of cancer and the various forms of treatment. Through the Committee on the Treatment of Malignant Diseases, under the Chairmanship of Dr. Robert B. Greenough, the College is in a position to report many five-year cancer cures. If additional cases can be recorded in the literature as proof that cancer is curable it will be advantageous to the profession and will lend great encouragement to the public by impressing upon both groups the importance of early and periodic advice based on accurate diagnosis.

COMMUNITY HEALTH MEETING

The people of St. Louis and vicinity will be given an opportunity to hear an unusually interesting program of brief instructive talks dealing with personal health and hospital matters at a Community Health Meeting to be held in the gymnasium of St. Louis University at 8 o'clock on Wednesday evening. A number of distinguished physicians and surgeons and leaders in the hospital field, from various parts of the country, have been invited to deliver addresses that will be illustrated in part by lantern slides and motion pictures. The program includes the presentation of a film on acute appendicitis created especially for exhibition to lay audiences.

HEADQUARTERS

General headquarters for the Clinical Congress will be established at the Jefferson Hotel, 12th and Locust streets, where the ballroom, Crystal and Ivory rooms and foyers adjacent thereto on the mezzanine and second floors have been reserved for the exclusive use of the Congress for scientific meetings, conferences, registration and ticket bureaus, bulletin boards, executive offices, scientific and technical exhibitions, etc. The ballroom of the Statler Hotel, at Washington and 9th streets, will be utilized daily for film exhibitions and scientific sessions on Tuesday and Thursday evenings.

TECHNICAL EXHIBITION

An interesting feature at headquarters will be the Technical Exhibition for which space has been reserved on the mezzanine floor including the Crystal and Ivory rooms and large foyers adjacent thereto. There will be represented in this exhibition the leading manufacturers of surgical instruments, X-ray apparatus, operating room lights, hospital apparatus and supplies of all kinds, ligatures, bandages, pharmaceuticals; publishers of medical books, etc.

HOSPITAL STANDARDIZATION CONFERENCE

For the fifteenth annual hospital standardization conference of the College, an interesting program of papers, round table conferences and practical demonstrations dealing with many of the problems related to the hospital standardization program of the College has been prepared. The conference opens on Monday morning at 10 o'clock in the ballroom of the Jefferson Hotel. On Tuesday morning, afternoon and evening sessions will be held in the Tuttle Memorial Auditorium directly across Locust Street from the hotel. The sessions on Wednesday morning and afternoon will be held in the same Auditorium. On Thursday a series of practical demonstrations will be given in certain of the St. Louis hospitals.

The program for this annual conference has been specially planned to interest surgeons, hospital trustees, executives, nurses, etc., and the College extends an invitation to attend this conference to all persons interested in the hospital field.

Monday, 10:00 A.M.-12:30 P.M.—Ballroom, Jefferson Hotel

ALLEN B. KANAVEL, M.D., Chicago; President, American College of Surgeons, presiding.

Address of Welcome. CURTIS H. LOHR, M.D., St. Louis.

Greetings from the President-Elect. J. BENTLEY SQUIER, M.D., New York.
Report of the Fifteenth Annual Hospital Standardization Survey and Announcement of 1932 List of Approved Hospitals. FRANKLIN H. MARTIN, M.D., Chicago.
The Standardized Hospital as a Medical Education Center for the Community Profession. ALLEN B. KANAVEL, M.D., Chicago.
Discussion. HORACE J. WHITACRE, M.D., Tacoma, Wash.
Medical and Hospital Economics. DANIEL CROSBY, M.D., Oakland, Calif.
Discussion. FREDERIC A. BESLEY, M.D., Waukegan, Ill.
How the Hospital Management and Medical Staff Can Co-operate in Reducing Mortality Rate of Appendicitis. JOHN O. BOWER, M.D., Philadelphia.
Discussion. GEORGE DAVID STEWART, M.D., New York.
Oxygen Therapy in Hospitals, Equipment and Management of Service. WILLIAM THALHIMER, M.D., Chicago.
Discussion. GEORGE W. CRILE, M.D., Cleveland.

Monday—2:00-5:00 P.M.—*Ballroom, Jefferson Hotel*

Pertinent Problems Affecting Hospitals and Their Solution—From a Nation-Wide Survey. E. MURIEL ANSCOMBE, R.N., St. Louis.
Discussion. W. HAMILTON CRAWFORD, Hattiesburg, Miss.
Economic Conditions as Affecting Canadian Hospitals and How These Are Being Met. ARTHUR J. SWANSON, Toronto.
Discussion. ROSS MILLAR, M.D., Ottawa, Canada.
Co-operation of Hospital Boards and Hospital Executives with Medical Staffs in the Diagnosis and Treatment of Cancer. BURTON J. LEE, M.D., New York.
Discussion. BOWMAN C. CROWELL, M.D., Chicago.
Fusing the Triple Viewpoints on Nursing—Doctors', Nurses', and Hospital Executives'. MARY M. ROBERTS, R.N., New York.
Minimum Standards for Schools of Nursing. REV. ALPHONSE M. SCHWITALLA, S.J., Ph.D., St. Louis.
Discussion. J. DEWEY LUTES, Chicago.

Tuesday—10:00 A.M.-*12:30* P.M.—*Tuttle Memorial Auditorium*

Symposium—Efficiency and Economics as Applied to:
The Clinical Laboratory. J. J. MOORE, M.D., Chicago.
The X-Ray Department. EDWARD H. SKINNER, M.D., Kansas City, Mo.
The Physical Therapy Department. JOHN S. COULTER, M.D., Chicago.
The Administration of Anæsthesia. JOSEPH MCNEARNEY, M.D., St. Louis.
The Administration of the Food Service. EUGENIA SHRADER, St. Louis.
The Handling of Surgical Dressings and Supplies. SISTER PHILOMENA, St. Louis.
General Discussion opened by E. E. KING, St. Louis.

Tuesday—2:00-5:00 P.M.—*Tuttle Memorial Auditorium*

Round Table Conference: Administrative, Professional, Economic, and Social Problems as Affecting Hospitals. Conducted by R. C. BUERKI, M.D., Madison, Wis.

Tuesday—8:00-10:00 P.M.—*Tuttle Memorial Auditorium*

Chairman's Remarks. PAUL H. FESLER, Chicago.
Greetings from the Hospital Trustees of St. Louis. AARON WALDHEIM, St. Louis.
Criteria to be Observed in Selecting the Governing Body of a Hospital. C. W. MUNGER, M.D., Valhalla, N.Y.
Responsibility of the Governing Body in Selecting the Superintendent. C. G. PARNALL, M.D., Rochester, N. Y.
Removing Hospitals from the Influence of Politics. JOHN A. MCNAMARA, Chicago.
Discussion. E. P. HOGAN, M.D., Birmingham, Ala.
How Hospital Trustees Can Keep Abreast with the Advances in Hospital Administration. MATTHEW O. FOLEY, Chicago.
General Discussion opened by REV. R. D. S. PUTNEY, St. Louis.

Wednesday—10:00 A.M.-*12:30* P.M.—*Tuttle Memorial Auditorium*

Handling of Communicable Diseases in Connection with a General Hospital. HENRY ROWLAND, Toronto.
Discussion. WALTER C. D. KIRCHNER, M.D., St. Louis.
The Individual Doctor's Responsibility for Clinical Records. WALTER F. COLE, M.D., Greensboro, N.C.
Discussion. DEWELL GANN, JR., M.D., Little Rock, Ark.
The Value and Scope of Medical Social Service Work in the Hospital. GRACE BEALS FERGUSON, St. Louis.
Discussion. ROBERT E. NEFF, Iowa City, Iowa.
How the Medical Social Worker Can Assist in the Present Economic Situation. RUTH LEWIS, St. Louis.
Discussion. BERYL B. ANSCOMBE, R.N., Kansas City, Mo.
The Rôle of the Social Worker in the Diagnosis and Treatment of Cancer. ELEANOR COCKERILL, St. Louis.
Discussion. FRANK L. RECTOR, M.D., Evanston, Ill.
General Discussion opened by B. A. WILKES, M.D., Cape Girardeau, Mo.

Wednesday—2:00-5:00 P.M.—*Tuttle Memorial Auditorium*

Round Table Conference: Administrative, Professional, Economic, and Social Problems as Affecting Hospitals. Conducted by ROBERT JOLLY, Houston.

Thursday—9:00-12:00 A.M. *and 2:00-5:00* P.M.

Discussions and demonstrations at St. Louis hospitals conducted by ROBERT JOLLY, Houston, Texas, and MALCOLM T. MACEACHERN, M.D., Chicago, assisted by superintendents and heads of departments of hospitals. Organization of the hospital, organization charts; admitting and discharging patients, demonstration of complete procedure; problems associated with clinical records, complete record systems; accounting and bookkeeping methods, simplified systems; nursing administration and nursing service; operating room management, detailed procedure in handling major operations; food service, various types of trays for general and special or therapeutic diet; management of obstetrical department, set-up for birth room; demonstration of handling supplies; organization and management of a central supply room; preparedness for emergencies in hospitals; demonstration of administrative conference.

ADVANCE REGISTRATION

Attendance at the St. Louis session will be limited to a number that can be comfortably accommodated at the clinics—the limit of attendance being based upon the result of a survey of the amphitheaters, operating rooms and laboratories in the hospitals and medical schools to determine their capacity for accommodating visitors. It will be necessary, therefore, for those who wish to

attend the Clinical Congress in St. Louis to register in advance.

Attendance at all clinics and demonstrations will be controlled by means of special clinic tickets, which plan provides an efficient means for the distribution of the visiting surgeons among the several clinics and insures against overcrowding, as the number of tickets issued for any clinic will be limited to the capacity of the room in which that clinic will be given.

A registration fee of $5.00 is required of each surgeon attending the annual Clinical Congress, such fees providing the funds with which to meet the expenses of the meeting. To each surgeon registering in advance a formal receipt for the registration fee is issued, which receipt is to be exchanged for a general admission card upon his registration at headquarters. This card, which is non-transferable, must be presented in order to secure clinic tickets and admission to the evening meetings.

REDUCED RAILWAY FARES

The railways of the United States and Canada have authorized reduced fares on account of the St. Louis session of the Clinical Congress so that the total fare for the round trip will be one and one-half the ordinary first-class one-way fare. To take advantage of the reduced rates it is necessary to pay the full one-way fare to St. Louis, procuring from the ticket agent when purchasing ticket, a "convention certificate," which certificate is to be presented at headquarters for the signature of the general manager of the Clinical Congress and the visé of a special agent of the railways. Upon presentation of a viséd certificate to the ticket agent in St. Louis not later than October 25, a ticket for the return journey by the same route as traveled to St. Louis may be purchased at one-half the one-way fare.

In the eastern, central, and southern states and eastern provinces of Canada tickets may be purchased between October 14 and 20; in other sections of the United States and Canada at earlier dates. The return journey must be completed within thirty days from date of sale of ticket to St. Louis.

The reduction in fares does not apply to Pullman fares nor to extra fares charged for passage on certain trains. Local railroad ticket agents will supply detailed information with regard to dates of sale, rates, routes, etc. Stop-overs on both the going and return journeys may be had within certain limits.

ST. LOUIS HOTELS AND THEIR RATES

	Minimum rates with bath	
	Single	Double
American, Market and Seventh Sts.....	$1.50	$2.50
American Annex, Market and Sixth Sts..	1.50	2.50
Chase, Lindell Blvd. at Forest Park.....	3.00	5.00
Claridge, Locust and Eighteenth Sts.....	2.00	3.00
Congress, Union Blvd. and Pershing Ave.	3.00	4.00
Coronado, Lindell Blvd. and Spring Ave.	3.00	4.50
Fairmont, Maryland and Euclid Aves....	2.50	3.50
Forest Park, W. Pine Blvd. and Euclid...	2.50	4.00
Gatesworth, Union and Lindell Blvds....	3.00	4.50
Jefferson, Twelfth Blvd. and Locust St...	3.00	4.00
Kings-Way, Kingshighway and W. Pine..	2.00	3.00
Robert E. Lee, Eighteenth and Pine Sts..	....	3.50
Lennox, Ninth and Washington Ave.....	3.50	5.00
Majestic, Eleventh and Pine Sts.....	2.00	3.00
Mark Twain, Eighth and Pine Sts.....	2.50	4.00
Marquette, Eighteenth and Washington..	2.00	3.00
Maryland, Ninth and Pine Sts.....	2.00	3.00
Mayfair, Eighth and St. Charles Sts.....	3.50	5.00
Melbourne, Grand Ave. and Lindell.....	2.50	4.00
Missouri, Locust and Eleventh Sts.....	2.00	3.00
Park Plaza, Kingshighway at Maryland..	4.00	5.00
Roosevelt, Delmar and Euclid Ave.....	2.50	4.00
Senate, Union Blvd. and Pershing Ave...	3.00	4.00
Statler, Ninth and Washington Sts.....	4.00	6.00
Warwick, Fifteenth and Locust Sts.....	2.50	4.00

Full fare must be paid from starting point to St. Louis, and it is essential that a "convention certificate" be obtained from the agent from whom the ticket is purchased. These certificates are to be signed by the general manager of the Clinical Congress and viséd by a special railroad agent at Clinical Congress headquarters on or before October 21. No reduction in railroad fares can be secured except in compliance with the regulations outlined and within the dates specified. It is important to note that the return trip must be made by the same route as that used in going to St. Louis and that the certificate must be deposited at headquarters during the meeting and return ticket purchased not later than October 25.

An exception to the above arrangement is to be noted in the case of persons traveling from points in certain far western states and British Columbia, who will be able to purchase round trip summer excursion tickets which will be on sale up to and including October 15 with a final return limit of October 31. The summer excursion fare is somewhat lower than the convention fare mentioned above, but is available only in certain of the far western states and British Columbia. Tickets sold at summer excursion rates permit traveling to St. Louis by way of a direct route and returning by way of another direct route with liberal stop-over privileges.

SYMPOSIA

TREATMENT OF FRACTURES

Wednesday, 2:30 p.m.—Ballroom, Jefferson Hotel

THE subject of fractures is one of perennial interest to the practicing physician and surgeon, and has an economic importance that is scarcely appreciated. The College has a committee under the chairmanship of Dr. Charles L. Scudder which has been working since 1922 to improve the treatment of fractures on this continent. Correct methods applied early after occurrence of the fracture will secure optimum results. Education of the laity on the subject of fractures, as well as the education of the medical student and the practitioner in his early years, has formed one of the objectives of this committee. An increase in the number and complexity of fractures is a penalty of mechanical progress and makes incumbent upon the profession adequate preparation to meet this unfortunate situation.

The College has taken cognizance of this situation and a symposium on fractures has been prepared as an important phase of this Clinical Congress. Fractures of individual bones will be discussed from the standpoints of diagnosis and treatment by members of the Committee on Fractures and other leaders in this field. The educational value of this symposium will be measured by its subsequent effect in the diminution of the period of disability and the increase in the completeness of restoration of function of those who suffer from fractures.

TEACHING OF SURGERY AND THE SURGICAL SPECIALTIES

Wednesday, 2:30 p.m.—Jefferson Hotel

BELIEVING that an excellent opportunity exists to arrive at a plan for the teaching of surgery which will be possible and satisfactory, a committee has been appointed by the American College of Surgeons to study undergraduate, graduate, and postgraduate teaching of surgery and the surgical specialties. The members of the committee are: Dr. Fred C. Zapffe, Chairman; Dr. Elliott C. Cutler, Dr. Irving S. Cutter, Dr. George J. Heuer, Dr. Alexander R. Munroe, and Dr. Allen O. Whipple.

A number of eminent teachers and clinicians of the United States and Canada have been asked to participate in a symposium on the subject to be presented on Wednesday afternoon. Meanwhile, the committee is soliciting the opinions of chiefs or heads of surgical departments in the undergraduate, graduate, and postgraduate medical schools. Based on these opinions, there will be formulated for consideration an outline of approved courses in surgery and the specialties that may be used in building courses in individual schools.

This is not an effort to standardize the teaching of surgery or the specialties. The reports will emphasize what the teachers of these subjects believe to be the best means of imparting fundamental principles, and of laying a sound foundation for future development. It is the underlying desire to arrive at the best and most effective training of the surgeon and the specialist of the future.

INDUSTRIAL MEDICINE AND TRAUMATIC SURGERY

Friday, 2:30 p.m.—Ballroom, Jefferson Hotel

THE care of the injured man ranks in importance with the care of those who are disabled through disease. Safety measures for the prevention of injury have been widely adopted by industry, but adequate organization for the care of those who do become ill or injured has not been provided in all industries. The College has conducted investigations and surveys in large areas of the United States to ascertain present medical conditions in industry and to inform employers of adequate methods. Some of the results of these surveys will be presented by the investigators at this symposium, which will be held under the auspices of the Board on Industrial Medicine and Traumatic Surgery, of which Dr. Frederic A. Besley is Chairman. Other subjects of importance in industrial medicine and in traumatic surgery in industry and in the non-industrial world will be included.

PRELIMINARY PROGRAM FOR EVENING MEETINGS

BALLROOM, JEFFERSON HOTEL

Presidential Meeting—Monday Evening

Invocation.

Address of Welcome: EVARTS A. GRAHAM, M.D., St. Louis, Chairman, Committee on Arrangements.

Introduction of Foreign Guests.

Address of Retiring President: Intangibles in Surgery. ALLEN B. KANAVEL, M.D., Chicago.

Inaugural Address: Fundamentals of Specialism. J. BENTLEY SQUIER, M.D., New York.

The John B. Murphy Oration in Surgery: Pillars of Surgery. SIR WILLIAM I. DE COURCY WHEELER, M.S., F.R.C.S.I., Dublin, Ireland.

Tuesday, Wednesday, and Thursday Evenings

Symposium on Surgery of the Large Bowel:

Diverticulitis of the Large Bowel. VERNON C. DAVID, M.D., Chicago.

The Hopeful Prognosis of Carcinoma of the Colon. FRED W. RANKIN, M.D., Rochester, Minn.

Gynecological Symposium:

The Results of Irradiation in the Treatment of Functional Uterine Bleeding. FLOYD E. KEENE, M.D., Philadelphia.

The Detection of Clinically Latent Cancer of the Cervix. WILLIAM P. GRAVES, M.D., Boston.

Fracture Oration: Fractures about the Elbow. PHILIP D. WILSON, M.D., Boston.

Oration: Industrial Medicine and Traumatic Surgery. FREDERIC A. BESLEY, M.D., Waukegan, Ill.

Inflammation. SIR GEORGE LENTHAL CHEATLE, K.C.B., C.V.O., F.R.C.S., London, England.

Bronchiectasis and Its Treatment by Lobectomy in One Stage. HAROLD BRUNN, M.D., San Francisco.

A Discussion of Some Principles Involved in the Pathology and Treatment of Empyema Thoracis. JOSEPH A. DANNA, M.D., New Orleans.

An Experimental and Clinical Study of the Use of Radium in the Brain. LOYAL DAVIS, M.D., and MAX CUTLER, M.D., Chicago.

Some Observations on Appendicitis: A Review of Four Thousand Appendectomies. J. M. T. FINNEY, JR., M.D., Baltimore.

Convocation—Friday Evening

Invocation.

Conferring of Fellowships.

Conferring of Honorary Fellowships.

Presidential Address. The American College of Surgeons—Twenty Years of Ambitious Effort. J. BENTLEY SQUIER, M.D., New York.

Fellowship Address: Some New Things in Physics. ROBERT ANDREWS MILLIKAN, Ph.D., LL.D., Sc.D., Nobel Laureate; Director, Norman Bridge Laboratory of Physics, and Chairman of the Executive Council, California Institute of Technology, Pasadena.

SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

Ballroom, Statler Hotel—Tuesday and Thursday Evenings

Highways and Byways in Ophthalmology. HANS BARKAN, M.D., San Francisco.

History and Development of the Operative Treatment of Facial Palsy. ARTHUR B. DUEL, M.D., New York.

Suppuration of the Petrous Apex in Relationship to Meningitis. WELLS P. EAGLETON, M.D., Newark, N. J.

PRELIMINARY CLINICAL PROGRAM

GENERAL SURGERY, GYNECOLOGY, OBSTETRICS, ORTHOPEDICS, UROLOGY, PROCTOLOGY, SURGICAL PATHOLOGY, ETC.

WASHINGTON UNIVERSITY MEDICAL SCHOOL

BARNES HOSPITAL

EVARTS A. GRAHAM, M. B. CLOPTON, A. O. FISHER, G. H. COPHER, W. H. COLE, DR. ALLEN, W. R. RAINEY, I. Y. OLCH, R. ELMAN and P. HEINBECKER—9, daily. General surgical operations.

ERNEST SACHS and ROLAND M. KLEMME—9, daily. Neurological surgery.

JOHN R. CAULK, D. K. ROSE, J. H. SANFORD, OTTO J. WILHELMI and V. R. DEAKIN—9, daily. Genito-urinary surgery.

VILRAY P. BLAIR, J. B. BROWN and W. G. HAMM—9, daily. Oral and plastic surgery.

J. A. KEY, ARCHER O'REILLY, J. W. STEWART, T. P. BROOKES and F. A. JOSTES—9, daily. Orthopedic operations.

H. S. CROSSEN, OTTO H. SCHWARZ, F. J. TAUSSIG, Q. U. NEWELL, C. D. O'KEEFE and R. J. CROSSEN—9, daily. Gynecological operations.

ST. LOUIS MATERNITY HOSPITAL

OTTO H. SCHWARZ, G. D. ROYSTON, F. P. MCNALLEY, T. K. BROWN and R. PADDOCK—9, daily. Obstetrical operations.

H. S. CROSSEN, OTTO H. SCHWARZ, G. D. ROYSTON, Q. U. NEWELL, F. P. MCNALLEY, O. S. KREBS, C. D. O'KEEFE, T. K. BROWN, C. R. WEGNER, R. PADDOCK, R. J. CROSSEN, M. A. ROBLEE and J. E. HOBBS—2, daily. Demonstration of obstetrical and gynecological cases and specimens; clinics on cancer of the uterus, sterility and electrocoagulation.

BARNES HOSPITAL, ST. LOUIS CHILDREN'S HOSPITAL, MALLINCKRODT RADIOLOGICAL INSTITUTE

Clinical Demonstrations Daily 9 and 2

ERNEST SACHS. Cases of brain tumors.

ROLAND KLEMME. Sympathectomy.

ERNEST SACHS and ROLAND KLEMME. Trigeminal neuralgia.

ERNEST SACHS and COBB PILCHER. Pathology of brain tumors.

VILRAY P. BLAIR and J. B. BROWN. Carcinoma about the mouth.

J. B. BROWN. Carcinoma of the larynx.

VILRAY P. BLAIR and I. Y. OLCH. Pathology of parotid tumors.

J. R. CAULK. (1) Transurethral prostatectomy; (2) use of the cautery punch with pathological studies of the removed tissue.

D. K. ROSE. (1) The relationship of intracystic pressure to the formation of diverticula of the bladder; (2) clinical application of the cystometer for measuring bladder pressures; (3) carcinoma of the kidney and hypernephroma.

H. L. WHITE. Mechanism of urinary excretion.

I. Y. OLCH. Pathology of carcinoma of the breast in relation to clinical features and mortalities.

J. ALBERT KEY. (1) Clinical and experimental observations on chronic arthritis; (2) internal derangements of the knee joint; (3) treatment of osteomyelitis with bacteriocidal ointment gauze.

J. ALBERT KEY and FRANKLIN WALTON. The effect of venous stasis on the healing of experimental fractures.

J. ALBERT KEY and ROBERT MOORE. The effect of sympathectomy on the healing of bone and cartilage.

GLOVER H. COPHER. (1) The treatment of fractures of the forearm; (2) reduction of dislocation of the semilunar bone.

ALEXIS HARTMANN. The use of "combined solution" in surgery.

R. C. MCCAHEE. A study of the cases of pyloric stenosis in the St. Louis Children's Hospital.

J. BRONFENBRENNER. (1) Discussion on the use of tetanus antitoxin; (2) clinical applications of bacteriophage.

ROBERT ELMAN. The treatment of surgical shock with particular reference to the use of acacia solutions.

WARREN R. RAINEY. Minor surgical procedures about the anus and rectum.

GLOVER H. COPHER. Surgical treatment of carcinoma of the colon and rectum.

H. A. BULGER and I. Y. OLCH. Clinical and pathological manifestations of diseases of parathyroid glands.

A. D. CARR, ROBERT F. PARKER and MARGARET SMITH. The clinical and pathological manifestations of tumors of the islands of Langerhans.

N. A. WOMACK and E. A. GRAHAM. The surgery of hypoglycemia.

E. A. GRAHAM. Estimating the risk in operations on the biliary tract.

SHERWOOD MOORE and LOUIS AITKEN. Technique of cholecystography.

SHERWOOD MOORE. Interpretation of cholecystograms.

DREW LUTEN. The clinical syndrome of coronary thrombosis in relation to upper abdominal pain.

JULIUS JENSEN. The evaluation of operative risk through a clinical study of the circulation.

H. L. ALEXANDER. Purpura in relation to abdominal pain.

D. P. BARR. Significance of pathological calcification.

D. P. BARR and LOUIS H. BEHRENS. Pituitary gigantism and dwarfism.

D. P. BARR and GLOVER H. COPHER. Milroy's disease, elephantiasis and the Kondoleon operation.

ROBERT EVANS. Modern treatment of syphilis, especially in relation to surgical problems.

WILLIAM H. OLMSTED. Clinical management of diabetic anteriosclerosis and gangrene.

I. Y. OLCH. Pathology of the blood vessels of the extremities in gangrene.

RALPH MUCKENFUSS. Diagnosis of fungus infections.

MCKIM MARRIOTT. Vitamins in clinical medicine.

LAWRENCE THOMPSON. Value of the Schilling hemogram in the study of acute surgical conditions.

J. F. BREDECK. The tuberculin test in the diagnosis of active tuberculous infection.

DR. LLOYD. Anomalies of renal veins and arteries.

GEORGE D. WILLIAMS. Anomalies of the recurrent laryngeal nerve with relation to the thyroid gland.

E. L. KEYES, JR. (1) Anomalies of the superior laryngeal nerve; (2) anomalies of the mesenteric attachment with relation to volvulus.

SHERWOOD MOORE and OSCAR ZINK. The value and limitations of X-ray therapy.

J. Hoy Sanford. Intravenous pyelography.
Sherwood Moore and D. K. Rose. Interpretations of pyelograms.
Hugh Wilson and Walter Seibel. Reduction of fractures under fluoroscopic control.
Sherwood Moore, Oscar Zink and Hugh Wilson. X-ray interpretation of chronic arthritis.
Charles O'Keefe. Hysterosalpingography.
J. W. Larimore. (1) Gastric and duodenal ulcer, etiology and treatment; (2) chronic duodenal diseases.
H. W. Wiese. Roentgenology of extra-alimentary tumors.
J. W. Larimore. Diagnosis of chronic appendicitis.
J. W. Larimore, Robert Evans and Charles Duden. Diagnosis of diseases of the cæcum.
John Caulk. Diagnosis and treatment of renal tuberculosis.
J. W. Larimore. Diagnosis and treatment of lesions of the œsophagus.
Sherwood Moore and M. F. Arbuckle. Diagnosis and treatment of foreign bodies in the respiratory tract.
E. A. Graham. Significance of intrathoracic negative pressures.
J. J. Singer, Alfred Goldman, Harry Ballon and Milton Smith. Diagnostic and therapeutic procedures of value in diseases of the lungs (pneumothorax, oleothorax, use of lipiodol, postural drainage).
Dr. Schurmen. Anomalies of origin and position of the phrenic nerve.
E. A. Graham, Duff S. Allen and J. J. Singer. Surgery in the treatment of pulmonary tuberculosis.
Paul D. Crimm (Boehne Hospital, Evansville, Ind.) and Clara Miller (Quincy, Ill.). After-care of the thoracoplasty patient.
Harry Ballon and H. A. McCordock. The mechanism of the development of tuberculous pneumonia following thoracoplasty.
E. E. Glenn. Atelectasis in pulmonary tuberculosis.
Kenneth Burdon, Philip Varney and Duff Allen. Etiology of lung abscess.
J. J. Singer, Duff Allen and E. A. Graham. Diagnosis and treatment of lung abscess.
J. J. Singer, Harry Ballon and E. A. Graham. Diagnosis and treatment of bronchiectasis.
E. A. Graham. Cautery pneumectomy for chronic pulmonary suppuration.
H. A. McCordock. Pathogenesis of brain abscess associated with pulmonary suppuration.
Duff Allen. Hemothorax, its treatment and relation to the production of empyema.
E. A. Graham. (1) Treatment of acute empyema; (2) treatment of chronic empyema.
Elsworth Smith, Duff Allen and E. A. Graham. Surgical treatment of heart disease.
Alfred Goldman. Sedimentation studies on pleural fluids.
J. V. Cooke. Mediastinal involvements of leukemia.
J. J. Singer, Harry Ballon and Herbert Carlson. Diagnosis and treatment of carcinoma of the lung.
J. J. Singer and Harry Ballon. Diagnosis and treatment of mediastinal tumors.
Herbert Carlson. Superior vena caval obstruction.
Harry Ballon and Hugh Wilson. The œsophagus, stomach and heart following unilateral phrenicectomy.
Harry Ballon, Herbert Carlson and E. A. Graham. The effect of phrenicectomy upon cough.
Herbert Carlson. Postoperative pulmonary complications.
Peter Heinbecker. The nervous regulation of respiration.
Warren H. Cole and Nathan Womack. (1) Experimental production of pathological changes in the thyroid gland typical of exophthalmic goiter; (2) repair in the thyroid gland; (3) effects of certain extracts on basal metabolism.
Warren H. Cole. Studies on liver function.
Robert Elman. (1) Value of gradual decompression of the obstructed intestine; (2) the rôle of the pyloris in the regulation of gastric acidity.
Robert Elman and E. A. Graham. Pathogenesis of the "strawberry" gall bladder.
Robert Elman and J. B. Taussig. Cholesterol function of the gall bladder and the formation of cholesterol gallstones.
Robert Elman and Warren H. Cole. Cause of death in acute portal obstruction.
I. Y. Olch. (1) The use of micro-incineration in the study of surgical pathological tissues; (2) studies of the liver glycogen in certain surgical diseases.
Peter Heinbecker. The sensory and motor nerve changes during spinal anæsthesia.
Glover H. Copher. (1) Selective distribution of portal blood in the liver; (2) effect of urinary bladder mucosa on osteogenesis in the dog.
J. Albert Key. Intra-articular anaphylaxis.
M. B. Clopton. Indications for and results in splenectomy.

ST. LOUIS UNIVERSITY MEDICAL SCHOOL

ST. MARY'S HOSPITAL

Tuesday

William T. Coughlin—9. Brain tumor; carcinoma of the breast.
John Stewart—9. Stomach and duodenal ulcer.
W. W. Graves and Leroy Sante—9. Brain tumor and duodenal ulcer.
Philip Hoffmann, Franklin Albrecht and Carl Vohs—2. Orthopedic clinic.

Wednesday

William Kerwin—9. Gynecological operations, prolapse of uterus, carcinoma of uterus, Cæsarean section.
Leroy Sante—9. The X-ray in gynecology.
William D. Collier—9. Demonstration of gynecological specimens.

Thursday

William E. Leighton—9. Cancer of the neck.
Louis Rassieur—9. Gall-bladder operations.
Ralph Kinsella and William D. Collier—9. Demonstration of gall-bladder cases.
C. E. Burford and Joseph Glenn—2. Nephropexy.

Friday

Carroll Smith—9. Goiter operation.
Charles Sherwin—9. Carcinoma of the breast.
Ralph A. Kinsella and William D. Collier—9. Goiter cases.
H. H. Kramolowsky and George H. Koenig—2. General surgical operations and demonstration of cases.

ST. MARY'S INFIRMARY

Tuesday

Louis Rassieur—9. Abdominal surgery.
Harvey S. McKay—10:30. Goiter clinic.

Wednesday

William T. Coughlin—9. Surgery of the head and neck.
Carroll Smith—9. Surgery of the colon and rectum.
Hyman I. Spector—2. Chest surgery.

Thursday

ROBERT D. ALEXANDER—9. Rectal surgery.
CHARLES F. SHERWIN—9. Breast surgery.
PHILIP HOFFMANN—2. Orthopedic surgery.

Friday

WILLIAM KERWIN—9. Gynecology.
WALTER E. HENNERICH—9. General surgery.

ST. JOHN'S HOSPITAL

Monday

Staff—2. Dry clinic, bone cases. A. P. BRIGGS: Bone development. A. E. HORWITZ and C. LINDEMAN: Parke's disease. LEO WILL: Fractures. JOSEPH PEDEN: X-ray demonstration of bone cases.
W. H. VOGT and associates—2. Obstetrical clinic.

Tuesday

BRANSFORD LEWIS, G. CARROLL, LEO BARTELS, C. D. PICKRELL, G. H. KOENIG, J. M. SCHATTYN and ROBERT F. HICKEY—9. Urological operations.
O. P. J. FALK and ANTHONY BRENNAN—9. Discussion of diagnostic and medical aspects of urological cases.
Staff—2. Dry clinic, diseases of the lungs. J. L. MARDER: Carcinoma of lungs. B. MCMAHON: Abscess of lungs. A. MCMAHON: Heart and lungs in surgical cases. GEORGE GAFNEY: Empyema.

Wednesday

L. M. RIORDAN, PERCY H. SWAHLEN, WILLIAM VOGT and M. WEIS—9. Gynecological operations.
Staff—9. General surgical operations. WILLIAM P. GLENNON: Gall-bladder surgery. J. MCHALE DEAN: Stomach and intestinal operations. I. H. BOEMER: Abdominal surgery. G. T. GAFNEY: Carcinoma of the breast. A. MCMAHON and J. J. HAMMOND: Discussion of diagnostic and medical aspects of these cases.
A. P. MUNSCH, J. MCH. DEAN, A. MCMAHON, O. P. J. FALK and I. H. BOEMER—2. Borderline medical and surgical cases.
C. H. NEILSON, F. KRAMER, J. MCFADDEN, W. P. GLENNON and H. N. ALLEN—2. Symposium on goiter.

Thursday

Staff—9. General surgical operations. BRANSFORD LEWIS: Urological operations. WILLIAM VOGT: Gynecological operations. J. MCH. DEAN: Stomach and intestinal operations. W. P. GLENNON: Goiter operation. W. K. MCINTYRE: Rectal operation. E. H. BOWDERN: Demonstration of anæsthesia methods and apparatus. A. P. MUNSCH and H. G. BRISTOW: Discussion of diagnostic and medical aspects of these cases.
Staff—2. Dry clinic. J. P. COSTELLO: Diagnosis of acute abdominal conditions in children. R. HYLAND: Traumatic surgery. J. MCFADDEN: Neurological aspects of traumatic surgery. W. GALLAGHER: Treatment of varicose ulcers. O. P. J. FALK and J. J. HAMMOND: Symposium on gall-bladder diseases. R. HYLAND: The acute surgical abdomen.

Friday

Staff—9. General surgical operations. P. H. SWAHLEN and H. J. RINGO: Gynecological operations. WILLIAM GALLAGHER: Abdominal operations. T. R. KENNEDY: General surgery. FRED BAILEY: Abdominal surgery. A. J. RAEMDONCK and R. F. BARNES: Discussion of diagnostic and medical aspects of these cases.
A. A. WERNER—2. Endocrine disturbance.
WILLIAM VOGT and J. A. HARDY—2. Ectopic gestation.
A. E. HORWITZ—2. Orthopedic surgery.

FIRMIN DESLOGE HOSPITAL

Tuesday

E. A. DOISY—9. Ovarian extracts.
E. L. SHRADER—9:15. Theelin and ovarian extracts in coagulation.
A. A. WERNER—9:30. The effect of theelin on castrates.
J. B. MITCHELL—9:50. The action of theelin and theelol on fowl.
G. O. BROUN and H. L. LANGE—10:10. Theelin and ovarian extracts in epilepsy.
W. D. COLLIER—10:30. The effect of theelin on the genital tract of the female white rat.

Wednesday

ALBERT KUNTZ—9. Autonomic nervous system in relation to surgery.
K. CHRISTIANSEN—9:30. The autonomic nervous system and special senses.
G. O. BROUN and A. P. BRIGGS—9:45. Studies in bile peritonitis.
R. A. KINSELLA—10:30. Bacterial endocarditis.

Thursday

A. B. HERTZMAN and F. E. FRANKE—9. Demonstration and discussion of cerebral circulation.
JOHN AUER—9:45. Studies on the contraction of fibrin and fibrinoid substances.
A. P. BRIGGS—10:05. Newer aspects of nephritis.
W. H. GRIFFITH—10:35. Food constitution in relation to food consumption (appetite).

Friday

ALBERT KUNTZ—9. Structural changes in the autonomic ganglia and ganglion cells associated with certain diseases.
PHILIP KATZMAN—9:30. Anterior pituitary hormone.
M. S. FLEISHER and L. R. JONES—9:50. Serum sickness in rabbits.
G. O. BROUN and W. F. HOLMES—10:10. Studies on pernicious anæmia.

MOUNT ST. ROSE SANITARIUM

Tuesday

Symposium on Medical and Surgical Aspects of Pulmonary Tuberculosis

C. L. BOISLINIERE—9. Diagnosis of pulmonary tuberculosis.
E. H. KESSLER—9:20. Roentgen findings in pulmonary tuberculosis.
ALPHONSE MCMAHON—9:40. Differential diagnosis of toxic thyroid and pulmonary tuberculosis.
A. C. HENSKE—10:30. Pneumothorax in pulmonary tuberculosis.
C. W. EHLERS—10:50. Oleothorax in pulmonary tuberculosis.
J. L. MUDD—11:10. Surgical treatment of pulmonary tuberculosis.

Wednesday

J. L. MUDD—9. Thoracoplasty and phrenicectomy.

Thursday

J. L. MUDD—9. Thoracoplasty and phrenicectomy.
J. L. MUDD—2:30. Exhibition of postoperative patients.

Friday

J. L. MUDD and C. W. EHLERS—9. Demonstration of pneumothorax, oleothorax and phrenicectomy cases.

ST. ANTHONY'S HOSPITAL

Tuesday

W. GAYLER—9. Gynecological clinic.
E. H. RUND—9. Hysterectomy.
J. E. FERRIS—10:30. Gall-bladder surgery.
REUBEN SMITH—10:30. Hernia operations.

Wednesday

NEIL MOORE and E. E. SEXTON—9. Diseases of the kidneys.
WILLIS YOUNG—10:30. Plastic surgery.
M. J. PULLIAM—10:30. Appendectomy.

Thursday

H. S. MCKAY, J. C. LYTER, M. J. PULLIAM, R. M. S. BARRETT and P. NEUN—9. Stomach and gall-bladder operations; consideration of medical and pathological aspects; choice of anæsthetics.

Friday

H. S. MCKAY, M. J. PULLIAM, R. M. S. BARRETT and P. NEUN—9. General surgical clinic; demonstration of pathological specimens, lantern slides.

MISSOURI BAPTIST HOSPITAL

Monday

C. H. SHUTT—2. General surgery.
J. S. YOUNG—2. Radiology.
M. L. KLINEFELTER—2. Demonstration of pathologic fractures.
GEORGE IVES—3. Cytologic study of cancer.
R. M. KLEMME—3. Neurosurgery.

Tuesday

E. L. DORSETT—9. Gynecological operations.
M. L. KLINEFELTER—9. Bone and joint surgery.
J. E. GLENN—9. Genito-urinary surgery.
H. TALBOTT—9. General surgery.
W. BARTLETT and W. BARTLETT, JR.—9. Goiter surgery.
R. J. CROSSEN—2. Gynecology.
D. K. ROSE—2. Genito-urinary clinic.
GEORGE IVES—2. Demonstration of method of blood transfusion.
W. E. WERNER—3. Goiter etiology.

Wednesday

C. H. SHUTT—9. General surgery.
M. L. KLINEFELTER—9. Bone and joint surgery.
C. E. BURFORD—9. Genito-urinary surgery.
J. B. BROWN—9. Plastic surgery.
W. BARTLETT, W. BARTLETT, JR. and J. C. LYTER—9. General surgery.
R. K. ANDREWS, O. H. CAMPBELL, C. E. GILLILAND, L. R. HEMPLEMAN, S. D. GRANT, and J. C. LYTER—2. Internists' symposium on surgical failures.
C. E. BURFORD—2. Genito-urinary surgery.

Thursday

R. S. KEIFFER—9. General surgery.
M. L. KLINEFELTER—9. Bone and joint surgery.
W. S. WIATT—9. General surgery.
D. K. ROSE—9. Genito-urinary surgery.
W. BARTLETT and W. BARTLETT, JR.—9. Goiter surgery.
S. I. SCHWAB and W. BARTLETT—2. Psychiatric aspects of surgery.
S. B. GRANT—2. The heart in goiter cases.
J. P. ATLHEIDE—2. Genito-urinary surgery.
J. B. BROWN—2. Industrial surgery.
J. P. MURPHY—3. The larynx in goiter cases.
J. S. YOUNG—3. Physiotherapy.

Friday

M. L. KLINEFELTER—9. Bone and joint surgery.
H. M. MOORE—9. General surgery.
Q. U. NEWELL—9. Gynecological operations.
R. M. KLEMME—9. Neurosurgical operations.
W. BARTLETT, JR.—9. General surgery.
W. L. CONRAD and H. F. D'OENCH—2. Dental surgery.
E. L. DORSETT—2. Gynecology.
W. BARTLETT, JR.—3. Chief safety factor in goiter surgery.

ST. LOUIS CITY HOSPITAL

Monday

W. H. VOGT, PERCY H. SWAHLEN, T. R. AYARS and W. J. HANS—2. Obstetrical clinic.

Tuesday

MAX W. MYER, CHARLES F. SHERWIN and HENRY HASSETT—9. General surgery.
W. J. DOYLE and J. J. LINK—9. General surgery.
FRANCIS REDER and THOMAS S. WIMBER—9. Industrial and traumatic surgery, dry clinic.
GRAYSON CARROLL, GEORGE H. KOENIG and CLARENCE MARTIN—2. Genito-urinary clinic.

Wednesday

EMMETT RUND, WILLIAM STUDE and S. A. WEINTRAUB—9. Gynecological clinic.
H. H. HELBING, C. W. GAERTNER, A. V. MARQUARDT and W. H. CLITHERO—9. Gynecological clinic.
JOHN W. STEWART, A. E. HORWITZ and E. L. MORSE—9. Fractures, dry clinic.

Thursday

JOHN W. STEWART and J. L. FERRIS—9. General surgery.
FRANCIS REDER, J. W. THOMPSON and ROLAND S. KIEFFER—9. General surgery.
FRANK J. TAINTER, WALTER C. G. KIRCHNER and W. J. DOYLE—9. Penetrating wounds of the chest and abdomen, dry clinic.
H. H. KRAMOLOWSKY and BENJAMIN F. MAY—2. Genito-urinary clinic.
H. G. LUND and P. N. DAVIS—2. Genito-urinary clinic.

Friday

FRANK J. TAINTER and W. J. GALLAGHER—9. General surgery.
THOMAS S. WIMBER and N. M. FREUND—9. General surgery.
CHARLES F. SHERWIN and LEROY SANTE—9. Surgical and radiological treatment of cancer, dry clinic.

ROBERT KOCH HOSPITAL

Wednesday

Staff—1. Dry clinic. H. I. SPECTOR: Diagnosis and treatment of pneumoconiosis complicated by pulmonary tuberculosis. DUFF S. ALLEN and GEORGE KETTLEKAMP: Surgical treatment of bilateral pulmonary tuberculosis; partial apical thoracoplasty versus phrenicectomy in pulmonary tuberculosis. A. E. HORWITZ: Subtrochanteric osteotomy for contracture hip deformity. GEORGE S. WILSON: Schilling blood count in respect to the surgical treatment of pulmonary tuberculosis. RALPH EHRLICH: Artificial pneumothorax in the treatment of pulmonary tuberculosis in the negro.

JEWISH HOSPITAL

Tuesday

ELLIS FISCHEL, ERNST JONAS and J. PROBSTEIN—9. General surgery.
SAMUEL NEWMAN—9. Rectal surgery.
H. EHRENFEST, F. J. TAUSSIG, S. A. WEINTRAUB, GROVER LIESE, S. F. ABRAMS and Dr. PATTON—2. Obstetrical clinic.
Drs. GREY and SOMOGYI—2. Demonstration and discussion of experimental work of surgical significance.

Wednesday

R. M. KLEMME—9. Neurosurgical clinic.
H. EHRENFEST, F. J. TAUSSIG, S. A. WEINTRAUB, GROVER LIESE, S. F. ABRAMS and Dr. PATTON—9. Gynecological operations.
Drs. SINGER, SIMON and FRANK—2. Medical and surgical thoracic clinic with demonstration of unusual X-ray films.

Thursday

MAX W. MYER, HARRY SANDPERL, E. V. M. MASTIN and E. K. DIXON—9. General surgery.
B. MAY, D. K. ROSE and MCCLURE YOUNG—9. Genito-urinary surgery.
Medical Staff—2. Pre-operative medical care of patients.
PAUL LOWENSTEIN and J. PROBSTEIN—3. Technique of injection of varicose veins.

Friday

ELLIS FISCHEL, WILLARD BARTLETT and PAUL LOWENSTEIN—9. General surgery.
F. H. ALBRECHT, FRED JOSTES and J. A. KEY—9. Orthopedic surgery.
S. GREY—2. Pathological demonstration.
B. MAY, D. K. ROSE and MCCLURE YOUNG—2. Urological dry clinic.
P. C. SCHNOEBELEN—3. X-ray demonstration of gastro-intestinal lesions.

SHRINERS' HOSPITAL

Tuesday

C. H. CREGO—9. Operative lengthening of tibia and fibula.
C. H. CREGO—2. Leg lengthening cases, end-results.

Wednesday

J. B. BROWN—9. End-results after split thickness skin grafts.
Staff—2. Orthopedic end-results.

Thursday

C. H. CREGO—9. Orthopedic operations.
J. A. KEY—2. Orthopedic clinic.

ST. LOUIS COUNTY HOSPITAL

Tuesday

F. A. JOSTES—9. Orthopedic clinic.

Wednesday

E. L. DORSETT—9. Gynecology.

Thursday

W. E. LEIGHTON—9. General surgery.

Friday

F. L. DAVIS—9. Genito-urinary surgery.

BARNARD FREE SKIN AND CANCER HOSPITAL

Tuesday

FRED J. TAUSSIG, S. S. LEVIN, E. S. AUER and FRED EMMERT—9. Surgery and radium therapy in cancer of the uterus and vulva.
FRED J. TAUSSIG, GEORGE GELLHORN, S. S. LEVIN, E. S. AUER, FRED EMMERT, KATE SPAIN and MARION WACHOWIAK—2. Malignancy index in gynecological cancer; technique of vulvar operations; exhibition of specimens.

Wednesday

ELLIS FISCHEL, CHARLES F. SHERWIN and GEORGE GAFNEY—9. Radical surgery and interstitial radium therapy.
D. P. BARR, C. M. STROUD and E. C. ERNST—2. Internal medicine and radiography in relation to cancer.

Thursday

GEORGE GELLHORN, S. S. LEVIN, E. S. AUER, FRED EMMERT, KATE SPAIN and MARION WACHOWIAK—9. Surgery and radium therapy in cancer of the uterus.
M. G. SEELIG, L. H. JORSTAD and E. C. ERNST—2. Demonstration of the production of tar cancer; pathological specimens, X-rays and photomicrographs of unusual problems in malignancy; specimens of crown gall in plants produced by bacillus tumefaciens; studies of mitochondria in cancer; reticulum in cancer growth.

Friday

W. E. LEIGHTON, GRAYSON CARROLL, THOMAS M. MARTIN and J. C. LANDREE—9. Surgical cancer therapy.
M. F. ENGMAN, RICHARD WEISS, A. H. CONRAD, C. V. LANE and M. F. ENGMAN, JR.—2. Amœbic and phagedenic ulcers and ulcers of unknown cause, presentation of cases, lantern slides.

ST. LUKE'S HOSPITAL

Tuesday

J. H. SANFORD, JOHN R. CAULK, OTTO WILHELMI, JOHN PATTON and C. E. BURFORD—9. Genito-urinary surgery.
D. STUTZMAN—10. Genito-urinary clinic.
J. H. SANFORD—11. Diagnosis and treatment of kidney lesions.
O. C. ZINK—11. X-ray interpretation.
R. M. KLEMME—11:30. Brain abscess.

Wednesday

C. D. O'KEEFE, OTTO KREBS, ROBERT CROSSEN and EDGAR SCHMITZ—9. Gynecological operations.
J. VAUGHN and GREY JONES—10. Obstetrical and gynecological clinic.
C. D. O'KEEFE—11. Ovarian cysts.
OTTO KREBS—11:30. Sterility.

Thursday

R. M. KLEMME—9. Neurological surgery.
A. O'REILLY and J. E. STEWART—9. Orthopedic surgery.
J. E. STEWART—10:30. Orthopedic clinic.
J. E. STEWART—11:30. Fractures of upper third of femur.
O. C. ZINK—11:30. X-ray demonstration.

Friday

O. R. SEVIN—9. General surgery.
L. KEYES, C. E. HYNDMAN, E. V. MASTIN and E. K. DIXON—10. General surgical clinic.

DePAUL HOSPITAL

Tuesday

EUGENE J. O'MALLEY and HENRY A. HASSETT—9. Surgical clinic, outpatient department.
FRANK TAINTER, ROBERT E. SCHLUETER and R. EMMET KANE—2. General surgical operations.
H. H. KRAMOLOWSKY—2. Genito-urinary clinic, moving pictures.
C. J. ALTHAUS—2. Genito-urinary operations.
ROGERS DEAKIN—2. Renal tuberculosis.
J. F. BREDICK—2. Schilling differential count in surgical diagnosis.
L. D. CADY—2. Postoperative neuroses.

Wednesday

J. W. THOMPSON, ARTHUR GUNDLACH, E. J. O'MALLEY and C. E. HYNDMAN—9. General surgical operations.
V. P. BLAIR, J. B. BROWN and W. S. HAMM—9. Plastic surgery.
H. W. SOPER—2. Diathermy in benign and malignant lesions of rectum.
OLIVER ABEL, JR.—2. Estimating the cardiac factor in surgical risk.
CHARLES EYERMAN—2. Allergy in surgical diagnosis.
D. B. FLAVAN—2. Chemical electrocardiography.
T. WISTER WHITE—2. Pneumococcus peritonitis in children; differential diagnosis for appendicitis.

Thursday

A. J. GETTINGER and H. S. LANGSDORF—9. General surgery.
C. A. STONE—9. Orthopedic surgery.
E. J. O'MALLEY and H. A. HASSETT—9. General surgical operations.
A. P. ROWLETTE—2. Experiments on pyloric function and gastric acidity.
W. C. CORSON—2. Effect of irradiated ergosterol on blood coagulation.
F. R. FINNEGAN—2. The effect of acidosis on cancer.
E. P. BUDDY—2. Clinical demonstration.
W. G. BECKE—2. Surgery and diabetes.

Friday

L. M. RIORDAN, R. J. CROSSEN, F. P. McNALLEY, L. E. PATTON, PERCY SWAHLEN and H. J. RINGO—9. Gynecological operations.
V. P. BLAIR, J. B. BROWN and W. S. HAMM—9. Plastic surgery.
L. D. CASSIDY—2. Differential diagnosis of lesions of colon.
J. W. THOMPSON—2. Carcinoma of colon, exhibition of cases.
F. R. FINNEGAN—2. Carcinoma of œsophagus, diagnosis and treatment.

FRISCO EMPLOYES' HOSPITAL

Wednesday

R. A. WOOLSEY—9. Back injuries and back conditions.

Thursday

R. A. WOOLSEY—9. General surgical operations.

U. S. VETERANS' HOSPITAL

Tuesday

S. L. FILKINS—9. General surgical clinic.
J. E. WHEELER—10. Orthopedic clinic.

LUTHERAN HOSPITAL

Monday

T. P. BROOKES—2. Dislocations of the cervical spine. complications; demonstration of cases, lantern slides and moving pictures.

Tuesday

H. L. NIETERT—9. General surgical operations.
J. L. HUTTON, V. KLOEPPER and F. DEMKO—9. General surgical operations.

Wednesday

R. E. SCHLUETER and H. P. THYM—9. General surgical operations.
H. A. HANSER, T. H. HANSER, and A. G. KLEIN—9. General surgical operations with spinal anæsthesia.
H. A. HANSER, T. H. HANSER, and A. G. KLEIN—2. Embolectomy, demonstration of cases.
E. W. SPINZIG—3. Roentgenological diagnosis of spontaneous and traumatic pneumoperitoneum.

Thursday

J. L. HUTTON, V. KLOEPPER and F. DEMKO—9. General surgical operations.
H. L. NIETERT—9. General surgical operations.
H. G. LUND and JAMES O'DOWD—9. Urological operations.

Friday

H. A. HANSER, T. H. HANSER, and A. G. KLEIN—9. General surgical operations, spinal anæsthesia.
R. E. SCHLUETER and H. P. THYM—9. General surgical operations.
G. O. GAUEN and E. A. VOGEL—9. Obstetrical operations.

MISSOURI PACIFIC HOSPITAL

Tuesday

O. B. ZEINERT and associates—9. General surgical operations.
W. P. ELMER and associates—9. Medical diagnostic clinic.

Wednesday

I. H. BOEMER and associates—9. Abdominal surgery.
W. K. MUELLER and associates—9. Roentgenological clinic.
H. J. SCHERCK and associates—9. Genito-urinary surgery.

Thursday

A. O. FISHER and associates—9. General surgical operations.
W. P. ELMER and associates—9. Medical diagnostic clinic.

Friday

O. B. ZEINERT and associates—9. General surgical operations.
W. K. MUELLER and associates—9. Roentgenological clinic.
J. H. SANFORD and associates—9. Genito-urinary surgery.

BETHESDA HOSPITAL

Tuesday

ROLAND HILL and B. W. KLIPPEL—9. General surgical operations.

Thursday

ROLAND HILL and B. W. KLIPPEL—9. General surgical operations.

DEACONESS HOSPITAL

Monday

HERMAN NIETERT, FRANCIS REDER, FRED BAILEY, JOHN C. MORFIT, ROBERT E. SCHLUETER and A. R. SHREFFLER—2. Medico-surgical dry clinics.

Tuesday

FRED W. BAILEY, WILLIAM H. NORTON, A. V. MARQUARDT, LEO A. WILL and J. EDGAR STEWART—9. General surgery and orthopedic operations.

A. R. SHREFFLER, EDWIN SCHISLER, M. L. KLINEFELTER, GUY SIMPSON, N. C. GAYLOR and DREW LUTEN—2. Medico-surgical clinical demonstrations.

Thursday

E. LEE DORSETT, N. C. GAYLOR, JOHN W. STEWART, FRED W. BAILEY, FRANCIS REDER and HERMAN NIETERT—9. General surgical and gynecological operations.

L. H. HEMPLEMAN, LEO BROOKS, CLAUDE PICKRELL, CHARLES A. STONE, JOHN C. MORFIT, M. F. ARBUCKLE and FRED C. SIMON—2. Clinical demonstrations.

U. S. MARINE HOSPITAL

Monday

J. E. SMITH—2. Clinical demonstration of abscess of lung.

Tuesday

W. M. JONES—10. General surgical operations.

W. L. COREY—2. Clinical demonstration of abdominal tumor with obstruction of transverse colon.

Thursday

W. M. JONES—10. General surgical operations.

J. T. DELOUGHERTY—2. Clinical demonstration of pyloric obstruction of stomach.

Friday

W. M. JONES—10. General surgical operations.

SURGERY OF THE EYE, EAR, NOSE, AND THROAT

WASHINGTON UNIVERSITY MEDICAL SCHOOL

BARNES HOSPITAL

Monday

FREDERICK O. SCHWARTZ—2. Ophthalmological operations, strabismus.

Tuesday

L. W. DEAN and staff—9. Ear, nose and throat conference.

M. F. ARBUCKLE and A. W. PROETZ—11. Otolaryngological operations.

MEYER WIENER—2. Ophthalmological operations.

Wednesday

L. W. DEAN and staff—9. Ear, nose and throat conference.

HARVEY J. HOWARD—2. Ophthalmological operations, demonstrating akinesia, scleroconjunctiva suture, intracapsular cataract extraction.

Thursday

L. W. DEAN and staff—9. Ear, nose and throat conference.

Friday

A. J. CONE, B. J. MCMAHON and WILLIAM L. HANSON—9. Otolaryngological operations.

J. B. COSTEN, L. J. BIRSNER and F. K. HANSEL—11. Otolaryngological operations.

H. ROMMEL HILDRETH—2. Plastic surgery of the eye.

McMILLAN HOSPITAL

Daily, 9:00 and 10:30

Staff—Clinical lectures and demonstrations.

LAWRENCE T. POST: Slit lamp demonstration.

WILLIAM E. SHAHAN: Physiological apparatus (including thermophore).

WILLIAM F. HARDY: Ocular muscles.

H. ROMMEL HILDRETH: Ultraviolet light therapy.

B. Y. ALVIS: Cylinder skiascopy.

M. HAYWARD POST: Advanced refraction technique.

FREDERICK E. WOODRUFF: Ophthalmoscopy.

MAX W. JACOBS: Ocular changes during pregnancy.

J. E. JENNINGS: Color vision tests.

ROY E. MASON: Industrial ophthalmology.

Monday

HOWARD C. KNAPP—2. Ocular tuberculosis clinic.

MEYER WIENER—2. Diagnostic eye clinic.

WILLIAM M. JAMES—3. Ocular syphilis clinic.

F. K HANSEL—2. Allergic clinic.

C. C. BUNCH—2. Hearing tests.

H. N. GLICK, HELEN GAGE, ALLEN POTTER, and L. C. BOEMER—3. Otolaryngological diagnostic clinic.

A. W. PROETZ—4. Demonstration of cases.

Tuesday

M. HAYWARD POST—2. Diagnostic eye clinic.

F. K. HANSEL—2. Allergic clinic.

C. C. BUNCH—2. Hearing tests.

H. N. GLICK, HELEN GAGE, ALLEN POTTER, L. C. BOEMER and GEORGE SAUNDERS—3. Otolaryngological diagnostic clinic.

I. Y. OLCH and CLIFFORD MENZIES—4. Demonstration of cases, pathology of ear, nose and throat.

Wednesday

HOWARD C. KNAPP—2. Ocular tuberculosis clinic.

WILLIAM E. SHAHAN—2. Diagnostic eye clinic.

WILLIAM M. JAMES—3. Ocular syphilis clinic.

F. K. HANSEL—2. Allergic clinic.

C. C. BUNCH—2. Hearing tests.

H. N. GLICK, HELEN GAGE, ALLEN POTTER, L. C. BOEMER and GEORGE SAUNDERS—3. Otolaryngological diagnostic clinic.

L. K. GUGGENHEIM and DOROTHY WOLFF—4. Demonstration of cases, embryology and anatomy of ear, nose and throat.

Thursday

WILLIAM F. HARDY—2. Diagnostic eye clinic.

HARVEY J. HOWARD—3. Clinical conference in ophthalmology.

F. K. HANSEL—2. Allergic clinic.

C. C. BUNCH—2. Hearing tests.

H. N. GLICK, HELEN GAGE, ALLEN POTTER, and L. C. BOEMER—3. Otolaryngological diagnostic clinic.

W. F. WENNER and P. R. NEMOURS—4. Demonstration of cases, physiology of ear, nose and throat.

Friday

HOWARD C. KNAPP—2. Ocular tuberculosis clinic.
LAWRENCE T. POST—2. Diagnostic eye clinic.
WILLIAM M. JAMES—3. Ocular syphilis clinic.
F. K. HANSEL—2. Allergic clinic.
C. C. BUNCH—2. Hearing tests.
H. N. GLICK, HELEN GAGE, ALLEN POTTER and L. C. BOEMER—3. Otolaryngological diagnostic clinic.

CHILDREN'S HOSPITAL

Tuesday

H. W. LYMAN, I. D. KELLEY, JR. and L. E. FREIMUTH—11. Otolaryngological operations.

Friday

G. E. HOURN and A. M. ALDEN—9. Otolaryngological operations.

OSCAR JOHNSON INSTITUTE

Staff—Daily, 9:00 and 10:30, Laboratory demonstrations.
HARVEY D. LAMB: Pathology of the eye.
WILLIAM M. JAMES: Conjunctival cytology.
H. ROMMEL HILDRETH: Anatomy of eye and orbit.
GEORGE H. BISHOP and B. HOWARD BARTLEY: Physiology of the eye.
PERCY W. COBB: Physiological optics.
CHARLOTTE WEIGHARD: Chemistry relating to ophthalmology.
ROSSLEENE A. HETLER: Nutrition relating to ophthalmology.
LOUIS A. JULIANELLE and MARION C. MORRIS: Bacteriology of the eye.
R. WENDELL HARRISON: Tissue culture of the eye.
Staff—Daily, 2:00. Laboratory demonstrations, otolaryngology.
GEORGE E. HOURN, LOUIS J. BIRSNER, JAMES B. COSTEN, HARRY N. GLICK, I. D. KELLEY, JR. and DOROTHY WOLFF: Anatomy.
W. F. WENNER and P. R. NEMOURS: Physiology.
CATHERINE BUHRMESTER: Chemistry.
EVELYN DIXON: Bacteriology.
L. W. DEAN: Cytology.
A. J. CONE: Temperature changes.
LOUIS K. GUGGENHEIM: Embryology.
ARTHUR W. PROETZ: Sinus study.
B. J. MCMAHON and CLIFFORD MENZIES: Pathological studies.

CENTRAL INSTITUTE FOR THE DEAF

Wednesday and Thursday

MAX A. GOLDSTEIN, JULIA M. CONNERY and Staff—10. Recent developments in the training of the deaf child; preschool deaf child; the first instruction in speech and lip-reading; conservation of residual hearing; a play by deaf children; the end products of training.
MILDRED A. MCGINNIS and staff—10. The operation of a clinic for the correction of defects in speech with demonstration of selected types of cases.
HELEN M. GEBHART and VIVIAN GROSS—10. Practical accomplishments in lip-reading.
R. LORENTE DE NO, HELEN F. SCHICK and MAX A. GOLDSTEIN—10. Some phases of special laboratory research in neuro-anatomy, phonetics, acoustics and psychology as applied at Central Institute for the Deaf.

ST. LOUIS UNIVERSITY MEDICAL SCHOOL

FIRMIN DESLOGE HOSPITAL

Tuesday

JOHN GREEN—2. Local use of epinephrin and epinephrin substitutes as adjuvants to miotics in the treatment of glaucoma simplex, with demonstrations.
J. F. HARDESTY—2:30. Immediate reduction of intra-ocular hypertension by constitutional treatment, with clinical demonstrations.
M. L. GREENE—3. Winged keratotomy with basal iridectomy for acute or chronic glaucome, Luedde operation, with demonstrations and review of results.
ALBERT KUNTZ—3:30. Fundamental principles in neurological and mechanical control of intra-ocular pressure.

Wednesday

W. H. LUEDDE—2. The new Bingham-Chandeysson supergiant magnet.
M. L. GREENE—2:15. The giant magnet in ophthalmic practice; experimental tests showing its wide range of power; suggestions for its use in laryngology and bronchoscopy.
W. E. LEIGHTON—3. Usefulness of giant magnet in general surgery with experimental demonstrations.
HUGO REIM—3:30. Ocular by products in industrial surgery.

Thursday

EUGENE T. SENSENEY—2. Otolaryngological operations.
LAURA LANE (by invitation)—2. Ocular tumors, with demonstrations from Wintersteiner collection.
J. M. KELLER and C. J. GISSY—2:30. Ocular trauma, with demonstrations from Wintersteiner collection.
JOSEPH MULZER (Heidelberg, Germany)—3. Sympathetic ophthalmia, with demonstrations from Wintersteiner collection.
CARL T. EBER—3:30. Moving picture demonstration: Cataract operation, Luedde technique for glaucoma operation.
W. H. LUEDDE—3:45. Successful destruction of intra-ocular pigmented new growth by localized controlled heat (Shahan thermaphore). Demonstration of unique case after four years.

Friday

L. C. DREWS—2. Serological control of retinitis pigmentosa; review of clinical evidence.
R. L. JONES and FRANCIS J. ROBBEN—2:30. Preparation and distribution of ocular extract for retinitis pigmentosa.
W. H. LUEDDE—3. Surgical significance of mechanical factors in ocular accommodation; mechanical factors in progressive myopia and their control; presentation of cases.
CARL T. EBER—3:30. Moving picture demonstration: Cataract operation, Luedde technique for glaucoma operation.

ST. MARY'S HOSPITAL

Monday

W. E. SAUER, S. B. WESTLAKE, R. H. MILLIGAN and C. O. BROWN—2. Otolaryngological operations.

WILLIAM H. LUEDDE, JOHN GREEN and associates—2. Ophthalmological clinic.
C. E. RICE—2. Surgical treatment of trachoma.

Tuesday

C. E. RICE—2. Surgical treatment of trachoma.

Wednesday

JOHN GREEN and associates—2. Ophthalmological clinic.

Thursday

W. E. SAUER, S. B. WESTLAKE, R. H. MILLIGAN and C. O. BROWN—2. Otolaryngological operations.

ST. ANTHONY'S HOSPITAL

Monday

F. G. A. BARDENHEIER—2. Otolaryngological operations.
C. J. GISSY—2. Ophthalmological clinic, operations and demonstration of cases.

Wednesday

J. M. KELLER—2. Ophthalmological clinic, operations and demonstration of cases.

Friday

F. G. A. BARDENHEIER—2. Otolaryngological operations.

ST. MARY'S INFIRMARY

Tuesday

WILLIAM E. SAUER—2. Aural surgery.

Wednesday

J. F. HARDESTY and associates—2. Ophthalmologica clinic.

ST. JOHN'S HOSPITAL

Tuesday

E. P. NORTH, V. L. JONES, N. R. DONNELL and JOHN MCGRATH—2. Demonstration of ophthalmological cases.
C. F. PFINGSTEN—2. Otolaryngological operations.

Thursday

V. V. WOOD—2. Demonstration of otolaryngological cases.

Friday

V. V. WOOD and ELMER SCHLUER—9. Otolaryngological operations.

ALEXIAN BROTHERS HOSPITAL

Monday

J. M. KELLER—3. Ophthalmological clinic.

Tuesday

D. P. FERRIS—2. Otolaryngological clinic.

Wednesday

C. J. GISSY—3. Ophthalmological clinic.

Thursday

D. P. FERRIS—2. Otolaryngological clinic.

MOUNT ST. ROSE SANITARIUM

Wednesday

WILLIAM SMIT—2:30. Otolaryngological clinic.

Friday

WILLIAM SMIT—2:30. Otolaryngological clinic.

DEPAUL HOSPITAL

Tuesday

V. V. WOOD—2. Otolaryngological operations.
L. J. BIRSNER—2. Anatomy of neck in relation to deep infections originating in throat and their surgical treatment.
W. P. DONOVAN—2. Otolaryngological operations.

Wednesday

T. P. LAWTON—2. Otolaryngological operations.
GEORGE HOURN—2. Otolaryngological operations.

Thursday

W. E. SAUER—2. Otolaryngological operations.
V. V. WOOD—2. Otolaryngological operations.
W. P. DONOVAN—2. Otolaryngological operations.
G. H. POOS—2. Ophthalmological operations.

LUTHERAN HOSPITAL

Tuesday

F. C. SIMON—2. Otolaryngological operations.
H. N. GLICK—3. Surgical consideration of structure of petrous pyramid; demonstration of specimen, lantern slides.

Wednesday

A. HOOSS—2. Eye operations.

Thursday

F. C. SIMON—2. Otolaryngological operations.
FREDERICK O. SCHWARTZ—2. Eye operations.

MISSOURI PACIFIC HOSPITAL

Tuesday

W. G. PATTON and associates—2. Otolaryngological operations.

Wednesday

EMMETT P. NORTH and VINCENT L. JONES—2. Ophthalmology, diagnostic and operative clinic.
S. B. WESTLAKE and associates—2. Otolaryngological operations.

Thursday

W. G. PATTON and associates—2. Otolaryngological operations.

Friday

W. G. PATTON and associates—2. Otolaryngological operations.

ST. LOUIS CITY HOSPITAL

Tuesday

CARL T. EBER—2. Ophthalmological operations.
E. LEE MYERS—2. Otolaryngological operations.

Friday

E. LEE MYERS—2. Otolaryngological operations.

FRISCO EMPLOYES' HOSPITAL

Wednesday

RICHARD J. PAYNE—2. Pulmonary lavage.
J. ELLIS JENNINGS—3. Practical tests for color blindness; several color blind persons will be examined.

JEWISH HOSPITAL

Monday

EUGENE T. SENSENEY—2. Radical mastoidectomy.
I. D. KELLEY, JR.—2. Direct vision adenectomy.
A. M. ALDEN—2. Classic closure of mastoid fistula.

Tuesday

MAX W. JACOBS and B. Y. ALVIS—2. Ophthalmological clinic, operations and demonstration of cases.

Wednesday

E. LEE MYERS and staff—2. Demonstration of bronchoscopy cases; laryngectomy.
C. E. EIMER—2. Direct laryngoscopy examination (Haslinger).
I. D. KELLEY, JR.—2. Lynch suspension.
M. D. PELZ, O. R. DOBBS and MAXWELL FINEBERG—2. Diagnostic clinic with demonstration of cases.

Thursday

MEYER WIENER—2. Ophthalmological operations.

Friday

LOUIS K. GUGGENHEIM—2. Demonstration of cases.
A. M. ALDEN—2. Snare and guillotine tonsillectomy and demonstration of ligation of bleeder; dacryorhinocystotomy.
S. B. WESTLAKE—2. Radical mastoidectomy.

MISSOURI BAPTIST HOSPITAL

Monday

R. J. PAYNE—2. Otolaryngological operations.
H. N. GLICK—2. Otolaryngological operations.

Wednesday

R. J. PAYNE—2. Otolaryngological operations.
H. N. GLICK—2. Otolaryngological operations.
J. F. HARDESTY—2. Ophthalmological operations.

ST. LOUIS COUNTY HOSPITAL

Monday

O. W. KOCH, J. B. COSTEN and A. M. ALDEN—2. Otolaryngological operations.

Wednesday

C. P. DYER, WILLIAM F. HARDY and JOHN MCGRATH—2. Ophthalmological operations and demonstration of cases.

Friday

JOHN GREEN and CARL BEISBARTH—2. Ophthalmological operations.

ST. LUKE'S HOSPITAL

Monday

W. E. SHAHAN—2. Ophthalmological operations.

Tuesday

B. J. MCMAHON—2. Otolaryngological operations.

Thursday

B. J. MCMAHON—2. Otolaryngological diagnostic clinic.

DEACONESS HOSPITAL

Monday

V. V. WOOD—2. Otoiaryngological clinic.
F. C. SIMON—2. Otolaryngological operations.

Wednesday

V. V. WOOD—2. Otolaryngological clinic.
F. C. SIMON—2. Otolaryngological operations.

U. S. VETERANS' HOSPITAL

Tuesday

P. H. FINOT—11. Ophthalmological and otolaryngological clinic.